The Journal of Obstetrics and Gynæcology of the British Empire

VOL. XXVI. JULY, 1914. No. 1.

The Ætiology of Eclampsia and Albuminuria and their relation to Accidental Hæmorrhage.

(An Anatomical and Experimental Investigation.)[1]

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THE study of eclampsia (and the albuminuria of pregnancy) can be carried out from two points of view, the *clinical* and *anatomical*, which concerns itself with a description and interpretation of the symptoms and the morbid changes present, and the *experimental*, in which an attempt is made to reproduce these symptoms and morbid changes in the lower animals. The investigations here recorded were, to begin with, carried out along clinico-anatomical lines. From these preliminary studies there soon emerged an experimental line of inquiry. As a matter of fact, this was entered upon as a necessary test of the validity of the conclusions to which the preliminary investigations had led.

Within recent years most of the investigations into the cause of eclampsia have issued from the belief that the placenta is the most likely source of a poison, which all are convinced is the cause of the disease. There can be no doubt that pregnancy is the cause of eclampsia and that the poison is, at any rate, in its ultimate origin to be traced to the child or to the placenta. That it is not the child is proved by its occurring in cases of hydatid mole, where there is no fœtus. This leads inevitably to the conclusion that the direct or indirect source of the poison resides in the chorionic elements. That the relationship between the uterine contents and the toxæmic states is a very direct one is shown by their frequent immediate cessation after the emptying of the uterus, or after the intra-uterine death of the fœtus. On the other hand, even when the child dies *in utero*, the toxæmia may persist until the child and placenta are delivered. It would in such cases seem clear that the death of the child (the macerated state of which at delivery testifies to the time when this death occurred) must, by the consequent thrombosis of the vessels, prevent any poison which might have been produced in it from being conveyed to the placenta. The placenta, therefore, in such a case, must be the source of the poison; and to my mind, the only interpretation legitimate under such circumstances is that,

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although the placenta usually becomes detached from the uterine wall when the child dies, it may in some cases remain fixed. When this is so, it will continue to pour into the blood-stream the toxic materials and thus perpetuate the condition which killed the infant. We thus see that an analysis of apparently irregular and irreconcilable data leads to a simple explanation.

It is impossible to gainsay the fact that there is an intimate and perfectly direct association between the uterine contents and the toxæmia. One fact, however, apparently inconsistent with such a view, is *post-partum* eclampsia. In about 20 per cent. of cases eclampsia occurs after labour. The great majority occur on the first day, and probably within a few hours after labour. No matter how we would explain these cases, we must inevitably start by admitting that something of a chemical nature has been left behind after the birth of the child and the placenta. We thus see that any experimental investigation into the cause of eclampsia must concern itself primarily with the attempt to isolate from the placenta (not from the child) some chemical substance that can reproduce the changes in animals.

Relation between Albuminuria and Eclampsia and Placental Disease.

It has for long been recognized that there exists a definite association between the toxæmic states and placental disease. On the other hand, infarcts of the placenta are not infrequent in cases where there is no sign of toxæmia. They are more likely to be present in the last two months of pregnancy, and Eden has suggested, with great reason, that they are to be looked upon as signs of senility in a short-lived organ. Their relative frequency has been differently stated by different authors. Meyer found them in 2 per cent. of 1,124 placentæ, Rossier in 17½ per cent. of 1,174, whilst Whitridge Williams found white infarcts, measuring 1 cm. or more in diameter, in 63 per cent. of 500 placentæ. Such figures, though exhibiting a wide margin of difference, leave no doubt regarding the relatively high frequency of the condition. On the other hand, there can be equally little doubt that these changes are associated in some special manner with the toxæmias, albuminuria and eclampsia. In Rossier's figures given above 54 of the women suffered from albuminuria, and, in these, infarcts were three times more common than where the urine was healthy. In Meyer's patients, where albumin and casts were present, infarcts were four times more frequent than where there was a normal urine. Fehling found them in 50 out of 91 albuminuric patients. "It is accordingly apparent," says Whitridge Williams, after stating these figures, "that the majority of investigators, who have busied themselves with the subject, believe that a marked relation exists between

albuminuria on the part of the mother and infarct formation in the placenta." The infarcts, which are of greatest importance in this respect, are those of the recent or red variety. It is with them that albuminuria is especially associated. White infarcts, as such, seem to possess little clinical importance. Out of 7 cases of eclamptic placentæ examined by myself, there were massive red or purple infarcts in 5, whilst out of 6 cases of severe albuminuria, there were similar structures in 3. That is, in 13 toxæmic placentæ there was recent placental disease, evident to the naked eye, in 8—*i.e.*, roughly 60 per cent.

"What is the meaning of this obvious association between placental infarction and the toxæmias?" is a question that has often been asked. Most workers admit that it is impossible to explain this relationship satisfactorily, or they attribute it to the action of the general toxæmia, of which the albuminuria is an evidence. For example, Eardley Holland, whilst admitting that infarcts are more frequent in albuminuria and eclampsia, says that "they may be looked upon as the result of a chronic toxæmia; as to their connexion with eclampsia, they are merely accompaniments, not consequences. The presence of these chronic degenerative changes in eclamptic placentæ has been investigated by Brindeau and Nattan-Larrier, who give them no special significance." Whitridge Williams, in a summary of the position, in so far as he could interpret it, said "marked infarct formation is not infrequently observed, and often results in the death or imperfect development of the fœtus. It is usually associated with albuminuria on the part of the mother, though at present we cannot account satisfactorily for the relation between them."

Placental disease of this kind has been usually looked upon merely as an accompaniment or as the result of the toxæmic state. So far as I know, no serious attempt has been made to regard the placental disease as the cause of the toxæmia, and this in spite of the fact that there is, in all the evidence which has accumulated through the years, not one single item which is logically inconsistent with such an interpretation.

It *is* true, then, that placental infarction may be present without any evident toxæmia. It is also true that there may be a severe toxic condition, and no evident change in the placenta. It is the very readiness with which the truth of these statements can be proved that has, in my belief, obscured the real nature of the relationship. To complete the statement of this relationship, where there is massive recent infarction there is always a toxæmia.

The meaning of this apparent paradox will become apparent when I say that, after I had been working at the subject for some time, studying the placentæ in their clinical relationships, I discovered that, *if there is an acute toxæmia ending rapidly in labour,*

the placenta may present evidence of disease, or it may look perfectly normal to the naked eye. If, on the other hand, the acute attack passes off and labour only supervenes, say ten or fourteen days after, we find extensive recent necrosis. Plate I, *fig.* 2, *is a good example of such a case. This important finding at once suggested that it is the recent autolytic changes in the affected organ that generate the poison.* As I shall show, this conclusion from anatomical data is completely supported by the experimental results. It is this that explains why, in an albuminuria which becomes established gradually and persists for some time, one is more likely to find marked placental disease. *It is just the comparatively slow involvement of the placenta that allows of the continuance of the pregnancy and the evolution of the infarcted regions. Where there is a sudden and extensive involvement of the placenta, the toxæmia is so fulminant that the pregnancy ends before any naked-eye changes in the placenta are produced.* The import of these statements will emerge in a clearer light later when the experimental work is entered upon.

It should be remembered that the placental organ is unique in the sense that, if a part of its substance undergoes necrotic changes, the degenerating patch is a massive poison focus bathed all round by circulating maternal blood. A ready access into the systemic circulation of any toxic material is thus allowed. There is no other region in the body where such a condition of affairs could obtain.

It is easy to understand that a comparatively small placental disease may occur without any untoward effect. Under these circumstances the absorption of toxic products from the dying patches is so gradual, and, at any one time, so small in amount that it is tolerated. Where, however, this normal and limited change becomes excessive there is a pouring into the maternal circulation of more poison than the system can safely deal with. Such an explanation accounts at once for the presence of the toxæmia, where the placental disease is marked. I contend that it is a much more logical one than to suppose that the toxæmia develops independently and acts upon the placenta by hurrying up and exaggerating a process that is so common in a smaller degree as to be almost normal. The one view is an explanation of a relationship, the other is meaningless, except as the statement of an extraordinary coincidence.

Mild degrees of albuminuria are common in pregnancy, and may well be due to those cases of comparatively small placental involvement. An explanation, on the lines I have laid down, accounts likewise for the chief time incidence of the toxæmic states we are considering being in the last two months of pregnancy, just the time when the senile changes in the placenta are most likely to be in evidence. It accounts also for the very frequent cessation of

the symptoms with the intra-uterine death of the child and the detachment of the placenta. This circumstance is due to the fact that the separation of the placenta withdraws the poison-laden foci from the maternal blood-stream, and the protective mechanism thus initiated is continued by the blocking of the open mouths of the maternal vessels by the thrombosis that quickly occurs.

The Cause of Placental Infarction.

1. *General Anatomical Considerations.*

The placenta is a temporary structure, and is so constructed that it can come away readily during labour. It is only so closely incorporated with the uterine wall that shearing on this wall is prevented. As a matter of fact, naked-eye examination shows that all that serves to bind placenta to uterus are a number of thin films of decidua. As I have shown in another place, there are, in the uterine mucosa, none, or practically none, of the supporting elements, such as muscle, elastic tissue, &c., which are found in other regions. Derived as it is from the mucosa the same applies to the decidua. The vessels lose these supporting coats as soon as, or nearly as soon as, they enter the placenta. This fact must also be considered in relation to the loose connexion which is necessarily present between placenta and uterine wall.

If we examine the placenta still attached to the uterine wall, it is found that the thin decidual films, which attach the placenta, bridge across large venous channels. In point of fact, the placenta seems to rest almost entirely on a greatly expanded blood lake, broken up into individual spaces by the bridging sheets. Even to the naked eye one can see these spaces opening on the one side through the placental surface into the intervillous space, and, on the other side, becoming continuous with the sinuses in the muscular wall of the uterus. On the placental side the blood spaces are lined by a thin layer of compact decidua. As we know, from microscopic examination, this thin layer is complete practically all over the placental surface. Where the vessels perforate the placenta it is absent, and, in addition, here and there, comparatively large tracts on the surface may be seen where the villi dip straight into the blood spaces. It is through such openings that Veit believes there may be a wholesale "deportation" of villi into the maternal blood-stream. The separation of the placenta is rendered easier by an interesting change which occurs in the decidua. This consists of an œdematous loosening of the tissues, and is often seen in separated placentæ just under the compact layer (fig. 1), as also in the decidual plugs that extend from this layer into the substance of the placenta.

This arrangement between placenta and uterus is ideal from the point of view of ease of separation, but it obviously also means a ready tendency to vascular disturbance. There is a very loose

organic binding, and to all intents and purposes the placenta merely rests against the uterine wall. When the child is expelled the retracting uterus grasps and expels the placenta, the soft decidual fixing strands offering practically no obstacle. During pregnancy there is nothing to disturb the placental fixation. It is known that the uterus contracts intermittently, but this can do nothing but empty the placenta of its blood. The liquor amnii will disseminate the tension equally over the uterine and placental surfaces, and it is likely that the constantly recurring intermittent uterine contraction, by expressing the placenta at definite intervals, leads to a heart-like, pumping action. The placental blood space is, in a sense, a greatly expanded maternal vessel. When a decidual vessel pours its blood into this a great diminution in velocity and force must take place, just as occurs when a river pours its waters into a lake. This circulating speed will become less and less as the small stream issuing from the vessel passes further and further into the placental blood lake and becomes wider and wider. The flowing force, therefore, in this space, derived from the *vis a tergo* of the general circulation, must be extremely low, and for an efficient passage of the maternal blood throughout and away from the placenta other factors must come into play. The recurring systole and diastole of the uterine muscle must, by exercising respectively a compression and a suction action, play an important part. We can imaginatively recognize the necessary existence of other factors. Thus, the villi are greedily absorbing fluid from the circumambient blood, and this must entail a continual mass movement of the blood in the space. Then, the taking up of oxygen and the other ingredients in solution in the blood, and the giving out of waste products, must mean a constant chemical alteration in the blood with constant diffusion currents backwards and forwards of the respective chemical ingredients in an effort to readjust the equilibrium.

II. *Changes Present in Localized Placental Death.*

Every person who has studied even a small number of placentæ is familiar with the so-called white and red infarcts. These consist of cubical, wedge-shaped, or irregular, sometimes sprawling, areas of yellowish or reddish tissue, which have lost the normal spongy appearance. They are firm in consistence. In the great majority of cases they lie with their base against the decidual surface. Sometimes an infarct may, at first sight, seem to lack this relation to the decidua, and to be embedded in the centre of the placenta or even to lie up against the chorion. In the majority of cases, however, on tracing the sections of the infarct, it is found to strike the decidua at a broad base. The oblique position of the infarcted region is what led to the erroneous first impression. In my experience, this decidual relation is so constant that it clearly must be considered as

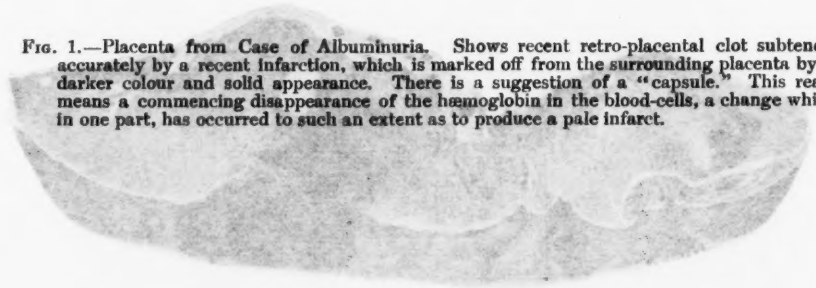

FIG. 1.—Placenta from Case of Albuminuria. Shows recent retro-placental clot subtended accurately by a recent infarction, which is marked off from the surrounding placenta by its darker colour and solid appearance. There is a suggestion of a "capsule." This really means a commencing disappearance of the hæmoglobin in the blood-cells, a change which, in one part, has occurred to such an extent as to produce a pale infarct.

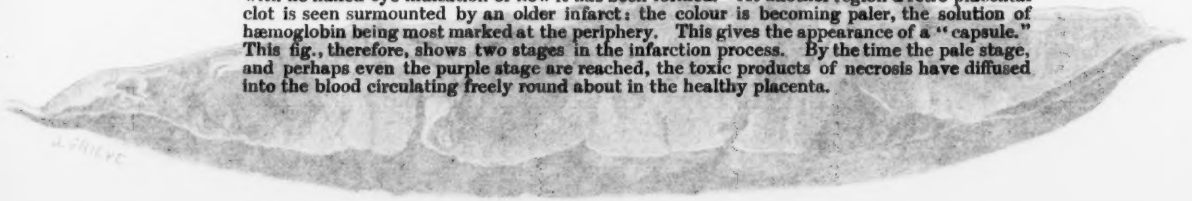

FIG. 2.—Placenta in Case of Eclampsia. In one part a large purple or recent infarct is seen, with no naked-eye indication of how it has been formed. At another region a retro-placental clot is seen surmounted by an older infarct: the colour is becoming paler, the solution of hæmoglobin being most marked at the periphery. This gives the appearance of a "capsule." This fig., therefore, shows two stages in the infarction process. By the time the pale stage, and perhaps even the purple stage are reached, the toxic products of necrosis have diffused into the blood circulating freely round about in the healthy placenta.

PLATE I.

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PLATE II

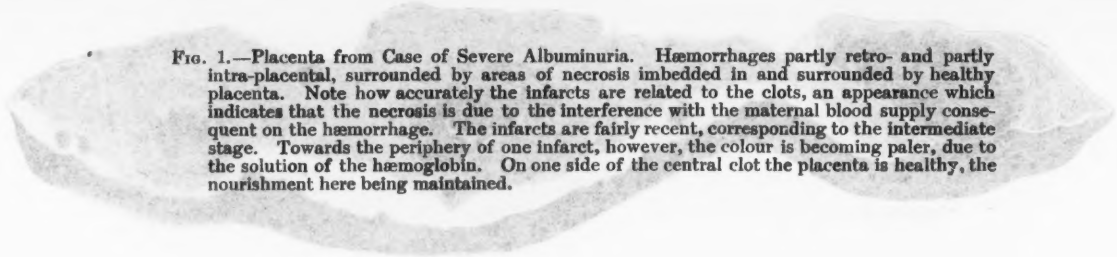


FIG. 1.—Placenta from Case of Severe Albuminuria. Hæmorrhages partly retro- and partly intra-placental, surrounded by areas of necrosis imbedded in and surrounded by healthy placenta. Note how accurately the infarcts are related to the clots, an appearance which indicates that the necrosis is due to the interference with the maternal blood supply consequent on the hæmorrhage. The infarcts are fairly recent, corresponding to the intermediate stage. Towards the periphery of one infarct, however, the colour is becoming paler, due to the solution of the hæmoglobin. On one side of the central clot the placenta is healthy, the nourishment here being maintained.

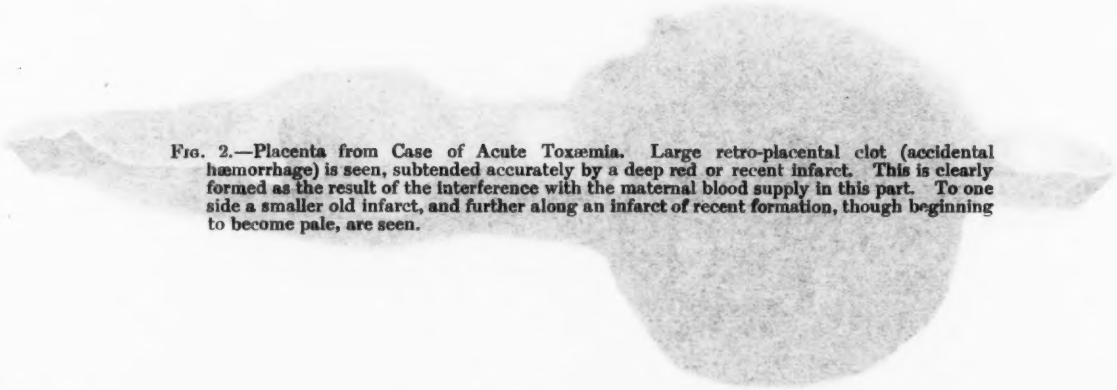


FIG. 2.—Placenta from Case of Acute Toxæmia. Large retro-placental clot (accidental hæmorrhage) is seen, subtended accurately by a deep red or recent infarct. This is clearly formed as the result of the interference with the maternal blood supply in this part. To one side a smaller old infarct, and further along an infarct of recent formation, though beginning to become pale, are seen.

PLATE II.

of great value and importance in any attempt to throw light on the causation of the condition. Many infarcts of the red or purple variety are difficult or impossible to recognize in the fresh placenta. When the placenta is put into fixing solution the blood quickly oozes from it, because in the healthy intervillous spaces it does not clot. (This it is which explains how rarely we find the villi embedded in blood in microscopic sections of placenta.) With the escape of blood the cut surface of the placenta, where it is healthy, becomes much paler. The diseased areas remain dark, and are thus thrown into relief against the paler background.

One fact, which has emerged clearly from my investigations is that the various appearances included under the term "infarct" are different stages in one and the same process and not, as is often stated in the text-books, independent pathological states.

(1) In the earliest stage recognizable to the naked eye the patch is deep red, purple or even black. *It may not be visible till thrown into relief against the surrounding paler placenta, which has lain for some days in the fixing solution, into which the unclotted blood in the intervillous spaces has oozed.* This stage is seen in Plate I, figs. 1 and 2, and Plate II, fig. 2. The patch stands out distinctly, and is sharply cut off from the surrounding, healthy placenta. In the very earliest stage this is all that is seen. When less recent, however, it is edged all round by a kind of capsule, seen on section as a thin whitish or yellowish streak (Plate I, fig. 1). Throughout the patch, black points and rounded spots are often visible, corresponding to blood in engorged villous vessels. In this stage the spongy character, though partly lost, has not quite gone.

(2) In this stage the colour becomes lighter. The dark red or purple becomes dusky brown, chocolate coloured or brick red. The spongy character of normal placental tissue has disappeared, and the patch cuts solid, the section showing a smooth surface all over (Plate II, fig. 1).

(3) In the older infarcts the colour becomes progressively lighter. The dark brown of the preceding stage becomes light brown, yellow, and then sometimes almost pure white.

That such a description of infarcts is the true one is proved by the fact that one can find, often in the same placenta, all the various stages between the earliest, purple, and the latest, white, stages. In Plate I, fig. 1, is shown a large infarct surmounting a large clot. That it is not quite recent is shown by the yellow "capsule," and by the fact that one part has passed into the pale stage. No doubt if it had been left the whole infarct would soon have become similar in appearance.

What is the explanation of these changing colours? Why is the later stage pale whilst the early is dark? The first explanation that suggests itself is that it is associated with an alteration in the blood

contained within the affected portion. That such is the case is proved by a microscopic examination of the various phases. To begin with, the villi are engorged throughout, the thin-walled capillaries in the small villi and the thicker vessels in the large villi are greatly expanded and are tightly packed with blood cells. In the small villi the vessel expansion may be so extreme that the stroma has become displaced to the periphery and there intervenes between the blood in the villi and that in the intervillous space only a very thin sheet of tissue (figs. 2 and 3). The purple colour of the fresh infarct is therefore due to the villi being turgid with poorly oxygenated blood. The whole appearance in this stage closely simulates the vascular hyperæmia and stagnation which occurs in other tissues when there is any local lowering in vitality, such as might result, for example, from injury. To my mind the engorgement, which is present in the early infarction, is of a smaller origin. It is an evidence of local reaction, which precedes the local death, that is inevitable when the blood supply of the part is cut off. In the later stages the blood within the villi, and any blood present in the intervillous regions, becomes paler and paler. The blood cells absorb the staining matter less and less. The hæmoglobin becomes completely removed and nothing but the shadowy outlines of the corpuscles are seen; or there may be a crumbling process, which results in a kind of granular debris—all that is left of the original blood cells. These stages correspond to the later stages of the infarction, and demonstrate readily the meaning of the phases presented by the changing nodule. It would thus seem that one of the chemical alterations occurring in the blood pent up in the vessels consists in a change in or solution of the hæmoglobin. Müller has shown that a similar change occurs during autolysis in the lung, and Mathes has reproduced the same change in the placenta by autolysis. In my experiments on placental autolysis, which I will record later, I have noticed the same result.

In association with these changes in the vessels and stroma there are other important structural alterations in the infarcted placenta. These have been carefully described by other investigators. One of the most frequent is a somewhat remarkable one. It consists of a *close packing of the villi* together throughout the whole or part of the infarct. In many cases the spaces between the villi are occupied, sometimes tightly, with blood cells. There may be at the same time a deposit of fibrin. This blood undergoes disintegrating changes similar to those already referred to in the villous blood. In the condition, however, which we are considering, the intervillous space is empty or nearly so, and the villi, instead of being separated from one another by comparatively wide intervals, as in the normal regions round about, have come close together. They may simply lie opposed to one another, or there may be an actual cohesion or

FIG. 1.

Decidual surface of placenta. Villi are seen above, then the thin compact decidua, and under this the loose decidua at the line of cleavage.

FIG. 2.

Recent (red) infarct. Villi packed together. Note the engorgement of the vessels. Case of accidental hæmorrhage without albuminuria.

FIG. 3.

Old (white) infarct. Villi packed together. They are degenerated and poorly staining. The expanded vessels are well seen. The blood cells in these cases are pale, due to a disappearance of the hæmoglobin. Case of albuminuria.

FIG. 4.

Edge of recent (red) infarct. To the right the densely packed villi are seen, to the left the healthy placenta. Case of albuminuria.

fusion of the epithelial surfaces (figs. 2, 3 and 4). In such cases the vessels are usually, as in other infarcts, greatly expanded, but not necessarily so, and the condition cannot be attributed to a general increase in the size of the villi, caused in this way, with a corresponding crowding of them together. Eden, so far as I know, first specially pointed out this rather remarkable condition, and explained it as due to a " progressive diminution of blood supply to a part of a cotyledon, by obliteration of a maternal artery [which] would cause the villi to become crowded together because there would not be sufficient blood in the part to maintain them at their normal distance from one another." The pressure exerted by the rest of the placenta " would drive the villi together into closely crowded, consolidated mass." This I believe to be the real explanation of the process. In other words, it must be looked upon as one of the possible changes where the local blood supply is disturbed. It will occur where the venous channels remain patent, or where the surrounding placental pressure can squeeze out the blood after obliteration of the afferent supply has taken place.

Clotting in the Intervillous Blood Space.—As I have already stated, one never finds any trace of coagulation in the intervillous space in the healthy placenta. In infarction a noteworthy appearance is the formation of fibrin, in the meshes of which red blood corpuscles are often entangled. *That this fibrinous deposit is not necessarily secondary to the changes in the villi is shown by the fact that we can often find it in conditions in which we definitely know there has been a stagnation of the maternal blood, and where the villi are healthy—e.g., accidental hæmorrhage.* As I shall show later the coagulation is primary; the necrosis, which occurs later in such cases, being secondary. As the result of the examination of a large number of placentæ, healthy and diseased, I have convinced myself of the truth of this statement. When we find a fibrinous deposit between the villi we can be sure that there is some grave interference with the maternal circulation in the corresponding part.

The *other changes* in the infarcted regions, which result in a gradual disintegration and softening, have been fully referred to by other writers, and need not be detailed here. The epithelial layers soon show changes. In the earliest stages a proliferation of the syncytial nuclei, forming masses of darkly staining tissue, would seem to be a sign found specially and characteristically in this condition. Later on both layers of the epithelium become pale and eventually crumble away. Similar changes attack the stroma and the cells of the vessel walls. In the later stages the autolytic process results in a solution of the tissues, with the result that cavities may be formed in the centre of the infarcts. The degenerative changes often seem to start and progress most rapidly towards the

centre of the nodules. In other cases they commence at the surface, as in the left-hand side of the infarct in Plate II, fig. 1, or in an eccentric position, as is shown in Plate I, fig. 1.

III. *Factors responsible for Infarction.*

Whilst an understanding of the exact origin of localized placental death is not essential to my interpretation of the toxic process, I believe that the ultimate explanation of albuminuria and eclampsia must remain hidden until light is thrown on these changes. There have, in the past, been two opposing ideas regarding the origin of placental infarction :—

(1) Eden, Whitridge Williams, and others believe that it is due to a change commencing primarily in the villi themselves. Their blood supply is interfered with as the result of an obliterating change in the vessels. This leads to a degeneration of the epithelium and stroma of the villi, and secondarily to a coagulation of the maternal blood.

(2) Other investigators, such as Hofmeier, believe that the degeneration is due to an interference in some way with the maternal blood supply.

The first, and, in this country, the most commonly accepted idea naturally assumes that the villi are dependent for their nourishment upon the fœtal blood supply, and that once this is obstructed they must undergo progressive necrosis. If one could show definitely that the villous structures are independent of the fœtal blood, and, moreover, can live and proliferate when this is removed, so long as the maternal supply remains intact, it would render this explanation of the infarction process untenable. This is not difficult to do. There are several considerations which show it :—

(1) The time when the chorionic elements are most active and proliferate most rapidly is during the early stages of the development of the ovum, where there are, as yet, no fœtal vessels formed, and where the trophoblast and its villi obviously live directly upon the mother's blood.

(2) In hydatid mole the chorionic villi live, and, as we know, actively proliferate, when there is not a trace of a fœtal vessel, and when the entire nourishment is derived from the blood of the mother. The same is true of chorionepithelioma.

(3) In tubal pregnancy one can sometimes recognize the independence of the villi of the fœtal blood in a diagrammatic manner. Where there has been a considerable hæmorrhage into the extra-chorionic space great masses of villi become strangled in blood-clot. I have seen one such case where all the trunks in the neighbourhood of the chorion had undergone fibrinous necrosis, but near the tube wall, where, in parts, the maternal circulation was unimpaired, the tips of the necrotic villi remained healthy (fig. 5).

These facts demonstrate beyond doubt that the villi, even after

FIG. 5.

Tubal pregnancy. Above, the necrotic villous trunks are seen strangled in blood-clot. Below, the tips of the same villi are seen to be healthy where they lie in spaces in which the maternal circulation is maintained.

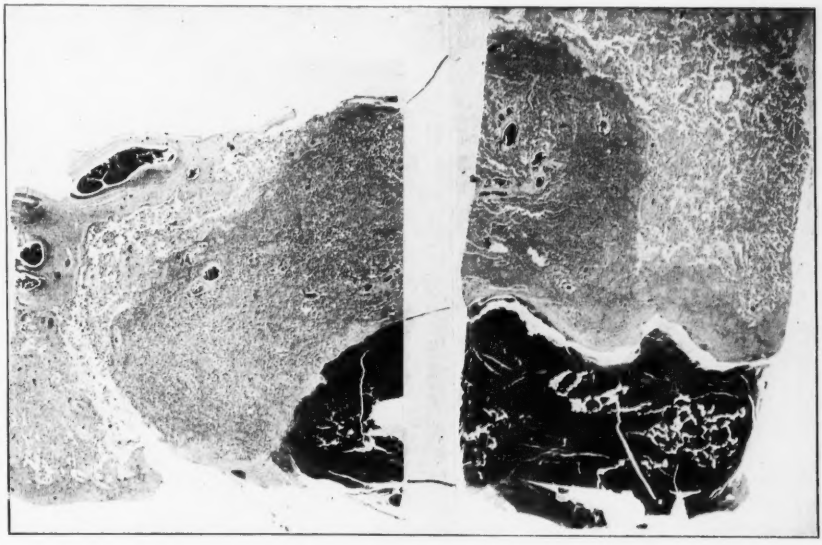


FIG. 6.

Recent (red) infarct accurately subtending retroplacental hæmorrhage. Shows dense packing of villi. The naked-eye appearance of this infarct is shown in Plate I, fig. 1. Case of albuminuria.

the fœtal blood supply is removed, can live, so long as the maternal supply remains uninvolved.

That the localized patches of dead tissue in the placenta are dependent upon an interference with the maternal blood supply is indicated by other findings of a positive nature:—

(1) As I have pointed out, in by far the largest number of cases, the infarcted areas lie in relation to the decidual surface. Moreover, in every such case one can detect degeneration, often necrosis, of the decidua. It is obvious that were infarction due to maternal causes, this is the disposition that would obtain.

(2) In large infarcts, resting upon the decidua, one sometimes can see, even with the naked eye, small nodules of healthy placenta on the outer or decidual aspect, which are obviously cut off entirely from the main part of the healthy placenta. Tracing in sections cut by the knife is sufficient to show this in such cases. This complete isolation of a healthy patch in a dead mass of tissue can mean one thing only, that whereas the remainder of the infarcted portion has lost its nourishment, the healthy nodule has retained its maternal supply. It is obvious that such a finding must be one of rare occurrence, but I have seen it twice, and in each case the healthy piece has rested against the decidua and into it healthy maternal vessels could be seen opening.

(3) *Retroplacental or Intraplacental Hæmorrhage as a Cause of Infarction.*—A consideration of the changes associated with placental hæmorrhage provides one of the most convincing proofs of the maternal origin of infarcts. Where there has been a hæmorrhage in the decidua serotina or into the placenta itself, there will be apt to be a local deprivation of the corresponding part of the placenta. The discovery of necrotic changes in these regions would be strong confirmatory evidence of the thesis I am trying to prove. As a matter of fact, it was the almost accidental discovery of this evidence that first convinced me of the fallacy of the usual explanation. One of the most surprising features of this investigation has been the great ease with which such evidence, and that of an unequivocal nature, has been obtained when it was looked for.

Where a retroplacental hæmorrhage is not quite recent one invariably finds the adjoining placenta diseased. The extent to which the necrotic process has gone depends upon the time during which the circulation has been cut off. Over old clots we see old, yellow, or white infarcts, whereas over young clots the infarction process is only in an early stage. Over clots of recent formation it may only be recognized on careful examination with the unaided eye, or it may not be evident without microscopic examination. In the earliest stages recognizable to the naked eye it consists of a deeply stained purple or dark brown patch, often difficult to recognize until the blood has been allowed to diffuse from the

adjacent healthy placenta. In Plate I, fig. 1, this appearance is shown. The necrotic area was at first difficult to recognize. It is seen to be not quite recent, because it is sharply outlined by a pale yellowish margin. Figs. 6, 7 and 9 show infarcts subtending retroplacental clots (see also Plate II, figs. 1 and 2).

In view of what I have said there can be little doubt that we are here brought face to face with an infarct in the making. The objection that might be urged, that the hæmorrhage is secondary to the necrosis, is disposed of by the appearance, a common one, seen in Plate II, fig. 1. Here the solid infarcted portions are seen closely and accurately nestling round the clot. If the necrosis had been primarily developed it is obvious that any bleeding into the placenta would not have chosen to so displace the solidified tissue. It would have found much less resistance in the loose retroplacental decidua.

As is well known, hæmorrhage in the placenta is commonly associated with toxæmic states. Not infrequently, in fact almost always, one finds masses of blood-clot of varying age adherent to the maternal surface. The possible ætiological importance of these clots, to which formerly little attention has been paid, will be evident from the statements just made, and this ætiological significance will be seen to force itself upon us even more clearly when we study the conditions that obtain in accidental hæmorrhage. The bleeding, in the cases we are dealing with, is most frequently retroplacental. Over the blood there is a thin layer of compact decidua, which is invariably necrotic, when the condition has been present for a sufficient time. In other cases the bleeding may be within the substance of the placenta. Here again such clots are often surrounded by a necrotic layer of placenta (Plate II, fig. 1).

Whilst the observations I have just recorded are amenable to one interpretation only and supply us with evidence proving that, in these cases, the necrotic placenta is dependent upon an involved maternal circulation, in many cases, in fact the majority, we do not find this unmistakable evidence. It is true that the majority of infarcts are related to the decidual surface, but in many it is impossible on naked-eye or microscopic examination to be sure of their exact mode of origin. The decidua is usually diseased and the vessels are thrombosed, but these changes might well be, considered in themselves, secondary, and have usually been so considered. A little thought, however, will show that a very likely explanation of the hæmorrhage in the decidua, to which I have called attention, would be blockage of the decidual veins or the veins in the muscular wall—*e.g.*, by thrombosis. This, in the presence of a free arterial supply, would tend to a backward pressure in the thin venous channels and would readily account for a hæmorrhagic escape, situated as they are in a soft unsupported decidua. Such an

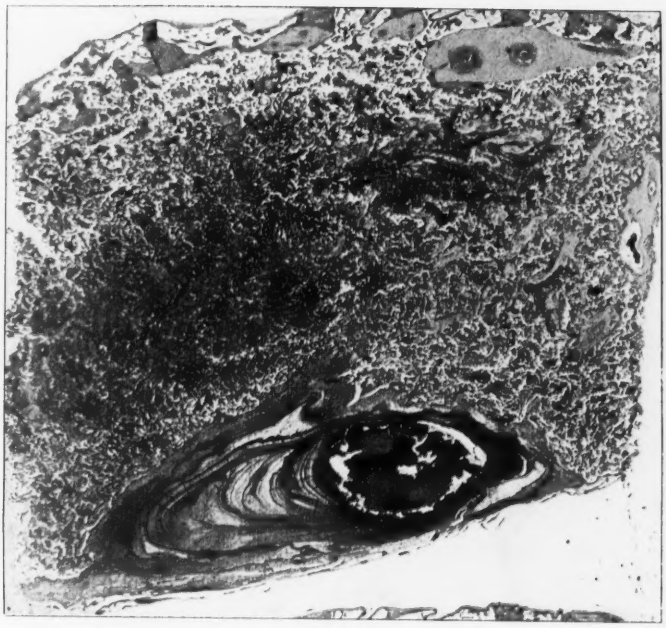


FIG. 7.

Recent infarct (invisible to naked eye in fresh placenta). The early stage of the consolidation process is seen in a region where the blood supply is cut off by thrombosis in the underlying vessel. Case of eclampsia.

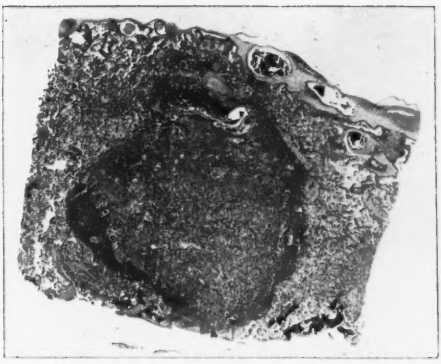


FIG. 8.

Recent (red) infarct. Shows consolidation process well advanced. In this there is no sign indicating how the maternal blood supply has been cut off. This information is often absent. Case of eclampsia.

FIG. 9.

Recent (red) infarct subtending retroplacental clot. This infarct was invisible to the naked eye. Case of severe accidental hæmorrhage without albuminuria.

explanation has been actually advanced by Veit as the cause of the retroplacental bleeding in accidental hæmorrhage, though he has attributed it to a blockage of the veins by masses of deported villi and not to thrombosis. Be this as it may, I believe that the almost certain factor operating in these cases is a blockage in the veins, and the frequency with which thrombosis is found in the decidual vessels and veins of the muscular coat suggests rather that the responsible agent is an excessive occurrence of this change, which is so frequent during the later months that it may almost be considered normal (Friedländer). Where the vein alone is blocked hæmorrhage will be the result. But this will only be one way in which the vascular changes will manifest themselves. *If there is a blockage of the artery, anæmia of the corresponding part of the placenta will occur* and this will be the only sign of the vascular involvement. That such is a very common factor in infarct formation is shown by the fact that a majority of the infarcts in my specimens show a close packing together of the villi into solid masses containing no blood in the intervillous space, *as if a kind of collapse has occurred subsequent to a cutting off of the entering blood*, just as collapse of the lung follows obstruction of a bronchus (figs. 6, 7, 8 and 9).

We may now summarize in a few words our conclusions regarding the origin of infarcts. In the first place, there can be no doubt that the chorionic elements are dependent immediately and directly upon the maternal blood for their nourishment, and can live and flourish where the fœtal blood is absent. This fact renders untenable the belief that infarction is due to an obliterating change in the vessels of the villi. A recognition of this fact leads inevitably to the conclusion that necrosis in the placenta will occur if the maternal blood supply is interfered with. I have shown that we can often find direct and undoubted evidence of such necrosis occurring in regions where there has been an arrest in the corresponding maternal supply of blood. Where there is an obstruction in the vein or veins hæmorrhage will be more likely to occur and will act as a signal post indicating the presence of the obstruction. Where the artery or arteries are involved, alone or in conjunction with the veins, anæmia of the part of the placenta concerned will occur followed again by necrosis. In such cases it may be difficult to find absolute anatomical proof of this mode of origin, but we may glean hints of its occurrence by a careful examination of the changes.

Remarks on the Maternal Circulation in the Placenta.

The observations I have just recorded indicate the fallacy of the usual idea regarding the circulation in the placenta. This is usually considered to be so free throughout, that any interference in one part is quickly made good by an inflow from the adjacent regions. The placenta is not to be looked upon as a loose sponge in which there is a sort of irregular mingling of the maternal blood. So far

is this from the truth that I believe it more likely that, just as in other tissues, each portion is supplied by certain maternal vessels and relies on them entirely for the nourishment. By injecting some colouring matter into the centre of an isolated lobule one can easily satisfy oneself of its isolation from the adjacent placenta, so far as its blood supply is concerned, for only with great force can we drive the fluid beyond it into the other parts. We can easily understand that this must be so, when we remember that, in the natural state, the fissures between the cotyledons are occupied by solid columns or partitions of decidua, which often run up throughout the entire thickness of the placenta. During separation these septa are left attached to the uterine wall. The separation is rendered easy in these regions by the œdematous loosening of the decidua immediately under the compact layer, which occurs in the later weeks of pregnancy, and to which I have already alluded. Not only are the cotyledons independent of one another so far as their vascular supply is concerned, but differing parts of the same cotyledon are dependent on different vessels for their nourishment. In each of these areas there is a complete circulation, though, at the boundaries of such circulatory systems, it is probable that there is a mingling of the bloods.

Relation between Accidental Hæmorrhage and the Toxæmias.

Accidental hæmorrhage is a subject of great importance in connexion with the thesis I am attempting to establish. The evidence which it provides is pertinent to the discussion and is even more convincing and less confusing than that to which attention has been directed on the preceding pages. In accidental hæmorrhage we have a retroplacental bleeding, which occurs with greater or less rapidity and leads to a corresponding amount of placental detachment. It must be considered as merely an exaggeration of the hæmorrhage in a similar site, which is fairly common in the last weeks of pregnancy. There are all grades between the smaller hæmorrhages (Plate I, fig. 1), through the comparatively large bleeding shown in Plate II, fig. 2, up to the condition we are considering, where there is an excessive loss of blood. The special interest which this condition possesses for us resides in its close relationship to the toxæmias, albuminuria and eclampsia. Its affinity with albuminuria has been recognized since it was pointed out many years ago by Winter. Its relation to eclampsia was referred to by Essen-Möller in a contribution to the recent International Congress in London. Essen-Möller reported two cases from his own clinic which were associated with eclampsia. Seitz has recorded two, Winter one, Harry one, and Hartmann two. Moreover, Bar and Kervily have found in one case, where there were no fits, degenerative lesions in the liver similar to those present

in eclampsia. (I quote these references to the literature from Berggren's paper.) Whilst this is so, one thing emerges clearly from the investigations on this relationship and is expressed by several of the authors—namely, that *it is impossible to look upon the toxæmia as the cause of the bleeding. For, in a large number of the cases, there is no trace of a toxic state.* For instance, in eight cases collated by Berggren, albuminuria was present only in four; in Zweifel's twenty-one cases there were thirteen cases without albuminuria. Essen-Möller brings out this same fact in a striking way. In 7,000 accouchements at the University of Lund, albuminuria was observed in 11 per cent., whilst accidental hæmorrhage was observed in only 0·45 per cent. He therefore says that "if albuminuria is a cause of retroplacental hæmorrhage, it is a cause of no great activity. But it is equally necessary to consider it from an opposite point of view. Out of my twenty-nine cases of accidental hæmorrhage, eleven showed albuminuria, and this figure rises to seventeen if we include cases with a trace only. So that whilst albuminuria is seen in only 11 per cent. of confinements, it is seen in 37 to 50 per cent. of cases of accidental hæmorrhage. It therefore seems to me that we can draw from these facts a second conclusion—namely, that the frequency of albuminuria in retroplacental hæmorrhage suggests something more than the idea of a coincidence pure and simple."

Such a condition of affairs, otherwise an admitted mystery, is readily explained in terms of the interpretation of the toxic states which I have already advanced. In accidental hæmorrhage we obviously have a corresponding part of the placenta cut off from its blood supply. If the hæmorrhage develops and extends so rapidly that it quickly kills the patient or determines a complete separation of the placenta, either naturally or by the interference necessitated in the patient's interests, there will be no opportunity for the necrotic changes to develop and there will be no toxæmia. The nature of the condition is such that termination in one or other of these ways must be common, and readily accounts for the fact that in about 50 per cent. of such cases there is no toxæmia. But, if the placenta remains attached at one part for some hours or days, the circulation there will remain undisturbed, and there will be an opportunity for the discharging into the maternal blood of the toxic ingredients, quickly elaborated by the disintegration of the separated portion. Only in such cases will an albuminuria or an eclampsia develop. As in the retroplacental hæmorrhages of smaller amount, so here it is easy, in cases where the blood-clot is not quite recent, to demonstrate the infarction changes in the severed portion or portions. The degree to which the change is manifest depends on the time that has elapsed since the bleeding occurred. I have had an opportunity in my specimens of recognizing only the earliest

stages. When present they are, however, distinct. On some occasions they have been found only after microscopic examination in a placenta that looked quite healthy to the naked eye. The discovery of these changes must be looked upon as strong confirmatory evidence in support of the truth of the reasoning which had foretold their existence. I shall refer, later on, to the question of the ætiology of accidental hæmorrhage, in so far as we have facts that permit of such an investigation.

I regret that a complete clinical history, so far as albuminuria is concerned, was obtained in none of my specimens. Whilst this would have given added interest to the discussion it is, however, not essential.

For the study of the anatomical changes in accidental hæmorrhage I have had one complete uterus, to which the placenta is still attached, and eight placentæ, as well as pieces removed from the uterine wall from four cases of fatal accidental hæmorrhage.

Changes in the Placenta.

The alteration in the placentæ varies with the age of the clot and the amount of separation that has occurred. In one case, in which there was not much hæmorrhage, there was no albuminuria. The labour ended soon after the first onset of the bleeding. In it an interesting appearance was found. The blood-clot was quite fresh. The placenta was examined soon after removal, and it was noted that the part of the placenta (about a quarter of the whole) that had been severed from the uterine wall was deeply congested as seen from the amniotic surface, the remainder being quite pale. This appearance corresponds to the earliest stage of the degenerative process, which is characterized by an expansion of the vessels in the villi. It would seem to be analogous to the congestion that occurs round an infarct in other parts of the body. The unaffected vessels undergo a rapid engorgement after the cutting off of the blood to the affected part. In the placenta the vessels of the villi, in which the circulation is primarily uninvolved, undergo a similar engorgement. For the rest, the degenerative changes in the placenta in cases of accidental hæmorrhage are similar to those which I have already described under the heading of infarction in general. In this condition, of course, they are more extensive, as large masses of the placenta, or even the whole placenta, are apt to be deprived of their blood supply. The changes may be equally developed in the affected parts or, where differing regions have been detached at different times, the changes may be more marked in some places than in others. One of the first changes to develop is a *fibrinous deposit in the intervillous space.* As I have already pointed out, this is to be looked upon as a certain sign of severe vascular involvement. It may be present before any visible changes have occurred in the chorionic elements. In

the majority of cases, however, where it is found there has occurred a congestion of the villous vessels (fig. 2).

In fig. 9 is seen another typical appearance found in these cases. A small recent clot is adherent to the decidual surface. Subtending this clot there is a region of definite consolidation in the placenta; in other words, a commencing infarct. It is localized to the region of the clot, and, even on naked-eye examination of the section, *the villi can be seen to be densely packed together,* the looser placental tissue all round being seen. Under higher magnification fibrinous threads are seen between the villi, and the vessels in the villi are greatly expanded. The epithelial covering of the villi is not obviously diseased. This section is taken from a placenta near a huge clot which had severed practically half of its surface from the uterine wall. As the result of the accumulating blood the placenta had been so compressed that its thickness was reduced by one-half, and in some places by two-thirds. Throughout this enormous piece of placenta changes similar to those just described were found—*i.e.,* there had taken place a huge early infarction.

At first sight it might seem possible to explain the degeneration in these cases as due to the compression of the blood-clot, and no doubt this must play a part in the devitalizing process. That it is not the necessary factor, however, is shown conclusively by the occurrence of the changes in the placentæ, in which a ready detachment has occurred without any obvious compression having taken place. This is, as a matter of fact, the usual condition of affairs. In these cases we find the intervillous coagulation and the congestion of the villi in the earlier stages, and the necrotic changes in the villi in the later stages. As I have said, a packing of the villi into solid masses, suggesting a collapse of the placental substance, is present in the majority of such cases. It is doubtless due to the anæmia of the part attendant on the interference with the blood entering, and, although the veins are almost certainly involved, and a leakage away along the normal channels is thus rendered impossible, the forcing of the blood out of the intervillous spaces into the surrounding, less involved, intervillous regions is probably a matter of comparative ease. Especially is this likely in view of the fact that, at the confines of the individual circulatory systems in the placenta there is probably under ordinary circumstances a certain mingling of the bloods. *In any case, the appearances leave no doubt of the fact that when the blood supply of any part of the placenta is interfered with, collapse and solidification is likely to occur.*

The appearances in the one case of accidental hæmorrhage in which I have been able to make a complete study of the placenta in situ are interesting. The placenta was completely separated by a huge blood-clot except at one edge. The hæmorrhage is completely

retroplacental, a fine layer of compact decidua covering the outer surface of the placenta. There were, throughout the separated part, changes such as I have referred to. The placenta is pale throughout, except at its attached edge, where the most recent disintegrative change is seen, in the shape of a large purple patch. It is clear that this is just the region where the most recent evidence of a degenerative process will be evident. It is the last part to be separated. As I have indicated on a preceding page, the purple colour soon gives place to a paler hue as the autolytic process leads to a solution of the hæmoglobin. This is the condition of the remaining part of the placenta. After an examination of such a specimen one can be left in no doubt whatever that the disease in the placenta is secondary to the deprivation of the maternal blood supply.

Changes in the Uterine Muscle, Decidua, and Vessels in Accidental Hæmorrhage.

The chief subject for this study has been the complete uterus, to which I have just referred. For this specimen I am indebted to Professor Kynoch, of Dundee. I have, in addition, been able to study sections cut from four complete uteri from fatal cases of accidental hæmorrhage. These are in the museum of the University of Liverpool, and, naturally, the chance of anything like a complete investigation of them was impossible. For them I am indebted to Professor Briggs. My remarks will be chiefly directed to the specimen of Professor Kynoch. In it a thorough examination was made. The other specimens served to provide confirmatory evidence of much that it demonstrated. The complete specimen consists of the uterus removed after death. The specimen was shown to the Edinburgh Obstetrical Society last year by Professor Kynoch, and I need not refer to the clinical history. The uterus is occupied by an immense blood-clot. The placenta has been detached by this, except along one edge, where it is still adherent to the uterus. There are marked and important changes in the muscular wall and in the decidua.

Changes in the Decidua.—These were discovered purely by accident as the result of the way in which the specimen was cut. The decidua vera is deeply congested. In the sections the *vera* is marked by a deep red line, and is obviously the seat of diffuse hæmorrhage or of vascular congestion. Microscopically the appearance was found to be due almost entirely to an enormous expansion of the decidual vessels into thin-walled sinuses. Here and there areas of hæmorrhage are present (fig. 10). In the adjoining muscular wall the vessels are in a state of congestion, and surrounding them there may be scattered hæmorrhages, but the changes here are nothing like so evident as in the decidua. The congested vera can be traced directly (even by the naked eye) into the serotina under the placenta, where the same vascular changes

FIG. 10.

Accidental hæmorrhage (Professor Kynoch's case). Piece of uterine wall beyond the placenta. To the left the amnion and chorion are seen, then the decidua vera showing greatly expanded vessels and hæmorrhage. To the right the muscular wall beset with small hæmorrhages. (It is therefore a case of "diffuse utero-placental apoplexy.")

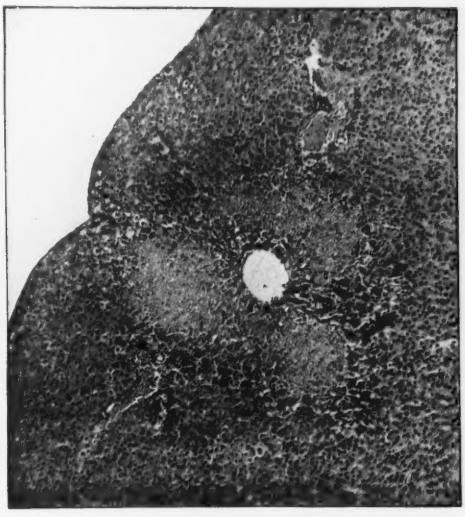


FIG. 11.

Liver of guinea-pig injected subcutaneously with extract of autolysed human placenta. Shows "focal necrosis" similar to that found in the human liver in eclampsia.

are present in an even more marked degree. In the *serotina* the vascular expansion is excessive, and there is an extensive blood leakage into the surrounding tissues. It is obvious that it is to the congestion and hæmorrhage here present that the enormous blood escape into the retroplacental space is due.

Changes in the Muscular Coat.—In no place is there any evidence of degeneration of the muscular tissue. In many places there is an expansion of the vessels, and an œdematous escape. In the muscular coat underlying the placental site (and also, but to a less extent, in that under the congested vera) the vascular changes are most evident, and here a hæmorrhagic leakage through the vessel walls has occurred. These appearances Essen-Möller and others have previously described. In one of my other specimens there was an extensive hæmorrhage into the muscular coat, which was visible as a deep purple patch under the peritoneum over the greater part of the anterior surface of the uterus.

It is evident, from a study of these changes, that the vascular disturbances present in accidental hæmorrhage are widespread, and in any attempt to account for them one must recognize that they are found throughout the uterus, and are not confined to the placental region. It has been amply proven that a toxæmic state cannot be considered as an ætiological factor, for in 50 per cent., if not more, there is no albuminuria. In discussing the causation of the milder degrees of retroplacental bleeding it was pointed out that a blockage in the veins of the decidua or adjacent muscle might quite well explain the condition. As a matter of fact, this explanation has been advanced by Veit to account for accidental hæmorrhage, his belief being that a mass of deported villi may plug the vein. In my belief this is an unlikely explanation, for it is difficult to see how a mass of villi sufficiently large could be severed from the placenta, though from Veit's sections which I have had the opportunity of examining, it is evident that there may occur a very luxuriant growth of villi along the veins adjacent to the placenta. To my mind a thrombosis in the veins is a more likely explanation. It is well known that thrombosis of the veins of the decidua and uterine muscle is so common that it may be looked upon as a normal change in the later weeks. It is also known that during pregnancy there is a very special tendency to the excessive formation of thrombi in the veins throughout the body. A blockage of the small veins near the placenta may quite well account for the smaller degrees of hæmorrhage in the retroplacental site. But the vascular changes present throughout the uterus in cases of accidental hæmorrhage are so widespread that it is evident that, if blockage of the veins is the cause, it must be a blockage that is far back in one of the main venous trunks in the uterus or even in the broad ligament.

As I have said, I had only one specimen in which a complete

examination of the changes was possible. In it I decided to investigate the main ovarian and uterine branches, and, to my surprise, without the least difficulty a massive, extensive, and fairly old-standing thrombosis was found in the ovarian vessels on each side, especially in the left. The uterine vessels seemed to be healthy. *It is therefore possible that accidental hæmorrhage is due to a thrombosis in a main venous trunk in the uterine wall, or even in the pelvis. The thrombosis would probably occur slowly, and might be present without any untoward signs, so long as it was not sufficient to obstruct the lumen of the vein completely. Let the obstruction become complete, especially let this occur suddenly, and the great venous pressure thrown back throughout the affected regions of the uterus would at once lead to the changes described.* It is impossible to make a dogmatic statement on the strength of only one case. This I do not wish to do, and merely describe the thrombosis in this case as a fact full of suggestion especially in view of the reasoning which led up to its discovery.

The main purpose of this investigation was not a discussion of the ætiology of placental infarction, or even of the ætiology of accidental hæmorrhage. I have devoted considerable space to their consideration, because I believe that the usual interpretation which is placed upon them has blocked the way for an enlightened effort to fathom the exact relationship existing between the toxæmic states (albuminuria and eclampsia) and placental disease. Hitherto this relationship has been considered as, in many respects, a mystery. This attitude, as I have tried to show, was due solely to the failure to recognize that placental disease must be considered as due to a cutting off of the maternal blood supply. *Whatever be the cause of the vascular interference, it is certainly not due to a toxæmic state. The study of the anatomy of placental infarction showed this, and the investigation of the conditions present in accidental hæmorrhage have proved it. It is clear, then, that if placental disease is especially prone to exhibit itself in association with a toxæmic state, and if it can be shown to be neither due to this toxæmic nor to any condition accompanying this toxæmic state, the only solution of the riddle is that the necrotic placenta is the source of the poison.*

There is no method of evading this conclusion. I pointed out early in this research that the clinical evidence in ordinary cases of albuminuria and eclampsia, taken in conjunction with the anatomical, strongly suggested that this was the true explanation of the relationship. An exactly parallel study carried out in regard to accidental hæmorrhage leads to the same conclusion.

Experimental Reproduction of Eclampsia in Lower Animals.

The anatomical investigations recorded in the preceding pages indicated that the poison or poisons responsible for albuminuria and

eclampsia are elaborated in a dying placenta. They, moreover, led to so precise a knowledge of the exact manner in which this process occurred that it seemed likely that an imitation of it *in vitro* should enable us to isolate the toxic material for experimental purposes.

One fact issued clearly from these investigations—namely, that the poison must be elaborated early in the course of the disintegrative change of the placenta; for there may be a fulminant toxæmia with an apparently healthy placenta. In severe toxæmias, however, if several days elapse before the birth of the placenta, massive, recently necrotic areas are visible. The time during which the placenta has been retained has allowed of the evolution of the devitalized patches into visible dark red or purple nodules of solidified tissue. In my earlier experiments this fact was not grasped. The placenta was autolyzed for eight days or longer, and, although a suggestion of success was every now and then obtained, the results were, on the whole, disappointing. With a clearer understanding of the exact process the results were positive all along the line. To begin with, a definite standard, by which the results were to be gauged, was formulated. Such a standard was not difficult to obtain, as the clinical and morbid picture in eclampsia is so precise. An attempt was to be made to reproduce this in lower animals in its totality. The main objects to be aimed at were: (1) the production of severe convulsions; (2) the reproduction of the liver changes, which, it is well known, are especially characteristic of eclampsia; and (3) the reproduction of the degenerative changes in the kidney.

Convulsions.—This, the main symptom in eclampsia, I had expected, when beginning my experiments, to find developing only some time after the animal had been brought under the influence of the extract of the autolyzed placenta; for it has been usually believed that the convulsive seizures are due to the accumulation in the system of some product of faulty metabolism, and are secondary to the liver or kidney involvement. When the proper method of preparing the placenta was discovered, one of the most immediate results of the injection (sometimes developing within twenty or thirty seconds) were severe and prolonged muscular spasms. In many cases the condition was an accurate reproduction of an eclamptic seizure.

Liver Changes.—These are so characteristic, as found in eclampsia, that I decided to make them the chief touchstone by which to test my results. Workers who have specially devoted their attention to these changes almost unanimously testify to their special association with eclampsia. They consist in a diffuse involvement of the organ. On naked-eye examination whitish areas are found scattered irregularly throughout its substance. In most cases small, they may in unusual cases form large white

patches. Microscopically, they are seen to consist of "focal necrosis," which, in the case of the younger and smaller patches, are especially distributed towards the periphery of the lobules. In addition, there is thrombosis in the vessels, especially towards the periphery of the lobules, and, in many cases, there are scattered hæmorrhages. So characteristic are these changes that Konstantinowitsch says that a diagnosis can be made from an examination of the liver alone. The specially characteristic feature is the localization of the early changes to the outer parts of the lobules. In this respect, also, my results have been positive. The subcutaneous injection of extracts made from autolysed placenta have given liver changes identical in every respect to those just referred to. I have been able to reproduce the focal necrosis, in the earlier stages located in the periphery of the lobules (figs. 11 and 12). In other cases, large, sprawling masses of necrosis have been found, and, in one animal, necrosis of a great part of one lobe of the liver was produced. Moreover, thrombosis in the vessels at the lobule periphery would seem to be the cause of the necrotic changes. Throughout the liver in these cases a diffuse fatty degeneration of liver cells is often present, as also scattered areas of hæmorrhage.

Kidneys.—The changes in the kidney, though usually found in eclampsia, are neither so consistent nor so characteristic as the liver changes. In many cases they are slight. In marked cases there may be patches of necrosis scattered throughout the organ. The part specially affected is the epithelium of the convoluted tubules, which shows a cloudy swelling, a fatty degeneration, or coagulation necrosis (Schmorl). In my experiments I have been able to reproduce these changes exactly, and it is especially important to note that the special region, which the toxic material that I have isolated affects, is the epithelium of the convoluted tubules (fig. 13).

Technique and General Method of Experiments.

The animals used were almost entirely guinea-pigs, and number forty-seven in all. For the extract fresh normal human placentæ were used.

Method of Preparation of Placenta.

To begin with, this caused considerable anxiety and trouble. As I soon discovered, it is well-nigh impossible to obtain a placenta in an absolutely sterile state. Some method had, therefore, to be discovered of preventing the growth of organisms, whilst allowing the natural ferments of the placenta to act. I first used thymol, adding a small piece of this to the saline solution in which the placenta was immersed. This method I employed when I was allowing the autolysis to continue for seven or eight days. I invariably found that there was a copious development of *Staphylococcus albus* by

that time. I next turned my attention to chloroform. This is admirable as an antiseptic, but I suspect that it must inhibit the action of the ferments. In some cases, however, I got a suggestion of positive results with this method. After the autolysis was completed the chloroform was extracted by evaporation *in vacuo*, and the juices were extracted by means of Büchner's press. The methods which I am at present using are three in number:—

(1) Once it was recognized that only a short period of autolysis was required it was thought that, by drying small pieces of placenta in the incubator, the drying process, whilst allowing a short autolysis, would take place so quickly that the risk of infection would be so meagre that it need not be allowed to enter into one's calculations. For this reason small pieces of placenta are washed freely in sterile water, spread out flat on sterile dishes, and placed in the incubator. In twelve or sixteen hours they are quite dry and can be powdered in a sterile mortar. The fibrous structure that remains at the end unpowdered can be easily removed by straining the whole through a fine wire sieve, previously passed through the flame. In this way we obtain a reddish-brown, fine powder that can be injected subcutaneously, suspended in saline solution.

(2) As the results obtained with this powder were so strikingly positive, it was decided to employ some other method of autolyzing the placenta, which would not be open to even the shred of suspicion, so far as infection is concerned, to which this powder may be held liable. For this reason pieces of placenta, about $\frac{1}{2}$ in. square, were washed free of blood as before, and placed in glycerine and incubated in it. When ready, two or three pieces are placed in a sterile mortar and pounded with 10 c.c. of saline solution.

(3) Recently I have been placing the placenta in saline solution, on the surface of which a film of toluol is placed. The proportions by weight of placenta to saline are two to one. If the whole is thoroughly shaken up before being placed in the incubator, enough of the toluol remains in solution to prevent any organismal growth. This method I began to use when it became apparent that the toxic material or materials liberated by the autolyzing placenta were extremely soluble. It was thought that by simply employing the supernatant solution into which the toxic substance had dissolved the same results should be obtainable.

Record of Experiments.

There are several points which require further investigation before a complete publication is possible. For example, I have obtained evidence, during the course of my work, that there are two distinct chemical substances responsible for the eclamptic attack. With the employment of the dry powder marked convul-

sive seizures are usually obtained, as also the liver changes. With the employment of the glycerine extract, whilst muscular spasms are often present, there are never convulsions, though the liver and kidney changes are obtained in a marked degree. These facts suggest that whilst the powder contains both of the toxic substances the glycerine extract contains chiefly that which has a special tendency to produce thrombosis. For these reasons I will record only those aspects of the results which are more or less final in their demonstration, and which are necessary to substantiate the belief that we have isolated the toxic material responsible for eclampsia.

Control Experiments.—A rigorous control was carried out. For this purpose twenty guinea-pigs (which were injected, sometimes over a long period, with extracts of the ovary or corpus luteum) were employed. These experiments were carried on coincidently with mine, and the animals were obtained from the same stock. For the opportunity of examining these control animals I am indebted to Dr. A. C. McMaster. In every case the liver and kidneys were examined. In none of them were convulsions, or other immediate signs of poisoning, ever produced. This disposed of the possibility that the spasms obtained in my experiments were merely due to the injection of foreign proteid, &c. In none of the controls were the liver and kidney changes discovered which are characteristic of eclampsia, and which I obtained with the placental injection. I think the absence of the typical liver lesions in such a large number of control animals may be considered as convincing proof of their specificity as found in my experiments.

Details of Experiments.

The injections were invariably made subcutaneously into the loose tissues of the back. By employing this method one had in view the avoidance of a rapidly fatal result, such as is apt to happen with intravenous injection. As other experimenters have shown (Weichardt, Pilz, Freund, &c.), the intravenous introduction of placental extract is quickly followed by death, due to a widespread intravascular thrombosis. Even though the right toxic substance had been isolated in their experiments, a proper test of its effect, as regards the morbid changes produced, was rendered impossible. Another objection, that of Lichtenstein, was overcome by the subcutaneous method of injection. This worker has shown that the intravenous injection of mineral matter in small particles—*e.g.*, sand—may cause results exactly similar to those obtained by Weichardt, Pilz, and Freund, and he believes that these were due, not to any toxic action on the part of the placenta, but merely to the introduction of free cellular elements into the circulation.

Injection of Fresh Placental Extract.—Five animals were

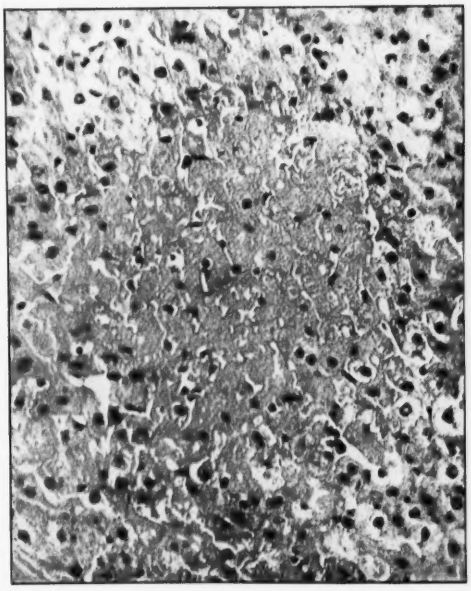


FIG. 12.

Liver of guinea-pig injected subcutaneously with extract of autolysed human placenta. Focal necrotic patch similar to that found in eclampsia.

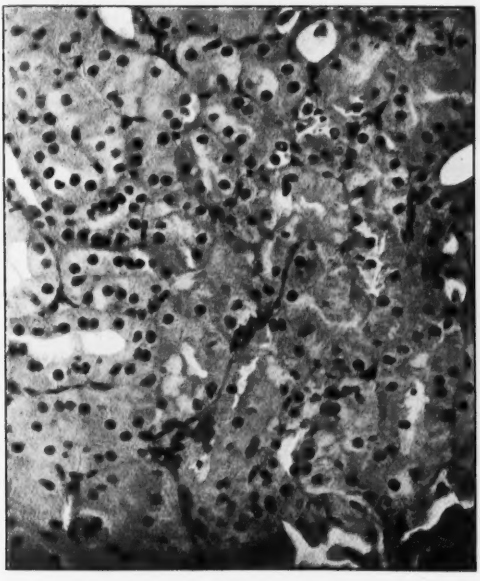


FIG. 13.

Kidney of guinea-pig injected subcutaneously with extract of autolysed human placenta. Convoluted tubules showing evident degenerative changes.

injected with the juice expressed from the placenta by means of Büchner's press. This is definitely toxic, if employed in large quantities, doubtless due to the action of a foreign proteid. In two animals 2 c.c., 3 c.c., and 10 c.c. were injected respectively on three successive days. The last dose killed within twelve hours. Three animals were injected with 10 c.c. of this extract at one time and two lived. In none were convulsions produced, and, in those that died no specific liver or kidney changes were observed.

Extract of Placenta autolyzed for Seven to Nine Days in Chloroform.—Thirteen animals were used: 5 to 8 c.c. of the extracted juice were injected once or twice daily till the animals died. In some this occurred after three to four injections, in others not till twelve or fourteen had been given. In one definite and marked convulsions were obtained; in most of the others general muscular spasms. Immediately after the injection the animals always became stupid and dull within a few minutes. In none were the liver and kidney changes produced.

Placental Powder.—It was the use of this that gave the first definitely positive results. Fifteen animals employed: 0·4 or 0·5 grm. of the powder was suspended in 6 or 8 c.c. of saline solution, and injected subcutaneously, usually night and morning. Three or four injections are sufficient to kill. In twelve, convulsive seizures were produced, varying from extremely marked tonic and clonic spasms of the whole body, lasting from ten minutes to an hour or longer, to general twitchings of the muscles of the head, trunk, and limbs. Other immediate signs of a toxic action were giddiness and a tendency to fall to the side, drowsiness with, in many cases, a complete absence of response on touching the eyes. To anyone acquainted with guinea-pigs these signs will at once assume suggestive proportions. In all, *degenerative lesions in the liver* were obtained, in six typical focal peripheral necrosis, whilst in two large, irregular sprawling masses of necrotic liver tissue were found. These appearances are shown in figs. 11 and 12. Degenerative lesions in the kidney, especially involving the convoluted tubules, were found, but these were neither so evident nor so constant as the liver changes (fig. 13).

Saline Solution in which Placenta was autolysed for varying short Periods of Time.—Six animals were used for this. Quantities varying from 5 to 10 c.c. of the supernatant fluid were removed after periods of autolysis varying from one and a half hours to sixty hours. These experiments were primarily carried out for the purpose of determining whether the toxic material passed readily into solution. For this purpose the presence of convulsive spasms was taken as a test. The results in this respect were positive, and showed that even as short a period of autolysis as one and a half hours was sufficient to liberate, at any rate, the element that provokes convul-

sions. The spasms are short-lived. Whereas, with the powder, the spasms may last for twenty minutes or longer, with this solution, even in markedly positive cases, the spasms rarely last for more than twenty or thirty seconds. So far, an investigation into the liver and kidney changes in such cases has not been carried out.

Placenta autolyzed for short Periods in Glycerine.—Eight animals were employed. The autolysis was allowed to continue for periods varying from ten to thirty-six hours. It soon became evident that the extract thus prepared is extremely toxic. Almost immediately after the injection, even of the extract of the short autolysis, the animals become dull and stupid, and often giddy. There may be general muscular spasms, but this is rare, a fact which shows that the specially toxic ingredient must differ from that present in the dried placental powder. Whereas with the powder the animals usually have recovered to a very large extent six or eight hours after the first dose, with this extract they never regain their usual vitality and remain till the end in a toxic state.

In this connexion it is interesting to note that the necrotic changes in the liver and kidney are especially marked with the glycerine extract. They were obtained in all the animals. In one animal the greater part of one lobe of the liver was found necrotic at the post mortem examination. The presence of well-marked kidney changes is important, in so far as they were not specially developed in the animals injected with placental powder. It is well known that, in eclampsia, the severity of the kidney lesions varies greatly in different cases. Even in fulminant cases they may be slightly marked. These considerations suggest that there are two different toxic agents operating in eclampsia and albuminuria, and that the exact clinical picture presented in an individual case, depends upon the relative proportions of these two materials present. The one material has a special affinity for the cerebrospinal tissues, and may be called the convulsive agent. The other has a special tendency to produce necrosis in liver and degeneration in kidney, perhaps, as has been suggested by many workers, because of a hæmagglutinative element.

Clinically, it is known that there are two different types of eclampsia. In one the chief symptom is convulsions, though it is usually associated with specially marked liver lesions and often presents kidney changes. In the other, there may be no convulsions. In these cases there would seem to be a special tendency for severe necrotic changes to occur in the liver and, especially, in the kidney. Under the latter category would come these cases of " symmetrical necrosis of the cortex of the kidneys," which Jardine, Teacher, and Kennedy, of Glasgow, and others have reported. These two different clinical types would correspond to the two different toxic bodies liberated by a dying placenta.

General Summary.

That eclampsia and the albuminuria of pregnancy are due to the liberation of the products of early autolysis of the placenta has been established by the following considerations:—

(1) The toxæmias are especially associated with recent infarction of the placenta. In severe cases, ending rapidly in labour, there may be no evidence, visible to the naked eye, of placental disease. If, however, the placenta is born several days after the attack, massive necrosis, obviously of recent origin, is seen. It requires some time for the necrosis to evolve into visible form.

(2) Placental infarction is due to an interference with the maternal blood supply of the part. It can be shown conclusively that the chorionic elements are dependent, immediately and directly, upon the maternal blood supply, and, so long as this is retained, can live, even when there is no fœtal supply.

(3) The interference with the blood supply, which is responsible for the infarction, is not dependent upon a toxic state and, in point of fact, may occur in the most extreme form, where there is no evidence of a toxæmia—*e.g.*, accidental hæmorrhage. An examination of the placenta, wherever there is definite evidence of an involvement of the maternal supply, invariably shows disease corresponding exactly to the area of this involvement. This disease will be evident to the naked eye unless the involvement is quite recent. The study of accidental hæmorrhage was shown to be specially important in this connexion.

(4) The placenta is so constructed that, if a part of it die, the products liberated from the dying patch can pass directly into the blood-stream. The organ is unique in this respect. It thus arises that for the occurrence of a toxæmia a circulation of blood round the poison-generating foci is necessary. An understanding of this fact at once dispels many of the difficulties associated with this study. It explains, for example, the cessation of symptoms after the death of the child (and separation of the placenta), and it explains the absence of a toxæmia in cases of accidental hæmorrhage (50 per cent. of the whole) in which the placenta is completely detached by the blood-clot, or by other means. The cases of accidental hæmorrhage associated with a toxæmia are those in which part of the placenta remains attached for some time after the separation of the adjacent part by a retroplacental bleeding. The necrosis of this part liberates the toxic materials.

(5) Where the placental disease is gradual in its onset there is more chance of the evolution of the infarcted patches. This explains why, in long-standing albuminuria, there may be more visible placental disease than in an acute eclampsia. It is just the gradual development of the toxæmia that allows of the pregnancy continuing.

(6) These facts all suggest that the toxæmias are due to the autolytic products liberated in the early stages of the placental death. By imitating the process, which occurs *in utero*, it has been possible to isolate from the healthy placenta a material or materials of a soluble kind which reproduce the clinical features and morbid changes, which all agree are especially characteristic of eclampsia. These are (*a*) convulsions, (*b*) peripheral focal necrosis in the liver, and (*c*) degenerative lesions in the kidney, especially located in the convoluted tubules.

In conclusion, I would say that, whilst post partum eclampsia constitutes the only difficulty which I know of in the way of absolute proof of my thesis, I think the evidence I have advanced is sufficiently cogent to necessitate a thorough exploration of the uterus in all such cases for a small piece of retained placenta. For a comparatively small piece may suffice to liberate fatal poisons.

I have to thank Sir Halliday Croom and Dr. Haultain for their kindness in allowing me to obtain the specimens of placenta from the Maternity Hospital during their period of office there. The work was carried out in the laboratory of the Royal College of Physicians, Edinburgh. To the Superintendent, Professor James Ritchie, I am indebted for kindly help. I have already expressed my thanks to Professor Kynoch of Dundee, Professor Briggs of Liverpool, and to Dr. A. C. McMaster, Edinburgh.

Bibliography.

Berggren. *Archiv mens. d'Obst. et de Gyn.*, Paris, 1912, ii, pp. 49-59.
Eden. *Journ. of Path and Bact.*, Camb., 1897, iv, p. 265.
Essen-Möller. *Archiv mens. d'Obst. et de Gyn.*, October, 1913.
Friedländer. Quoted from Eden.
Hofmeier. " Die menschliche Placenta," Wiesb., 1890.
Holland, E. *Journ. of Obst. and Gyn. Brit. Emp.*, Lond., 1909, xvi, pp. 255, 325, 384. (An excellent critical review, to which I am especially indebted for much of suggestion and fact.) Names not mentioned in this list are quoted from this paper.
Jardine and Teacher. *Journ. of Path. and Bact.*, Camb., 1911, xv, pp. 137-46.
Jardine and Kennedy. *Trans. Edinb. Obst. Soc.*, 1912-13, xxxviii, pp. 158-215.
Kynoch. *Trans. Edinb. Obst. Soc.*, 1912-13, xxxviii, p. 189.
Lichtenstein. *Archiv f. Gyn.*, Berl., 1908, lxxxvi, pp. 434-504; *Zentralbl. f. Gyn.*, Leipz., 1909, xxxiii, pp. 265,275, 1313-1325.
Mathes. *Zentralbl. f. Gyn.*, Leipz., 1901, xxv, 1385-89.
Müller. Quoted by Mathes.
Schmorl. " Path. Anat. Untersuchungen zur Pathogenese der Eclampsie," Leipz., 1893; also *Archiv f. Gyn.*, Berl., 1902, lxv, pp. 503-29.
Veit. " Die Verschleppung der Chorionzotten," Wiesb., 1905.
Whitridge Williams. *Amer. Journ. Obst.*, New York, 1900, xli, p. 775.
Winter. *Zeitschr. f. Geb. u. Gyn.*, Stuttg., 1885, xi, p. 398.
Young. *Proc. Roy. Soc. Med.*, Lond., 1911, iv (Obst. Sect.), pp. 291-306.
Zweifel. " Festschrift Internat. Gyn. Kongress," Berl., 1912, p. 55.

TECHNICAL MEMORANDA.

A Further Modification of Anterior Colporrhaphy combined with Amputation of the Cervix.*

By W. E. Fothergill, M.A., B.Sc., M.D.

Hon. Surgeon St. Mary's Hospital for Women and Hon. Assistant Gynæcological Surgeon, Royal Infirmary, Manchester; Lecturer in Obstetrics, University of Manchester.

Soon after I brought up this subject at our Leeds meeting in April, 1913, I again made a change in my method of removing the anterior vaginal fornix and the cervix by one single incision. My reason for mentioning the matter again so soon is that I should like those who may be induced to try the operation in cases of prolapse with hypertrophy of the cervix to have the advantage of my last year's experience.

It may be remembered that my first step in operating for prolapse was the extension of the usual anterior colporrhaphy wound widely into the lateral vaginal fornices so as to expose the connective tissue which lies on either side of the cervix—the parametric tissue, in short. The closing of this wound from side to side brings together in front of the cervix structures which previously lay at its sides, and so pushes the cervix back and up towards the hollow of the sacrum. The usual anterior colporrhaphy only exposes tissue at the sides of the vagina, and the closure of the incision from side to side leaves the uterus in retroversion. Amputation of the cervix by the usual methods shortens the anterior vaginal walls and pulls the uterus downwards and forwards. It is thus a bad preliminary to anterior oolporrhaphy. My next step, therefore, was to remove the cervix and part of the vaginal wall by one incision and to close this so as to push the cervix upward and backward. The steps of the operation, as dscribed last year, were as follows:—

1. Dilate the cervix.
2. Make a circular incision round the cervix.
3. Snip round the cervix and push up the bladder and parametria until a sufficient length of the cervix is exposed.
4. Incise the vaginal wall outward for an inch or more to either side of the circular incision.
5. Separate the anterior vaginal wall from the parametria and bladder.

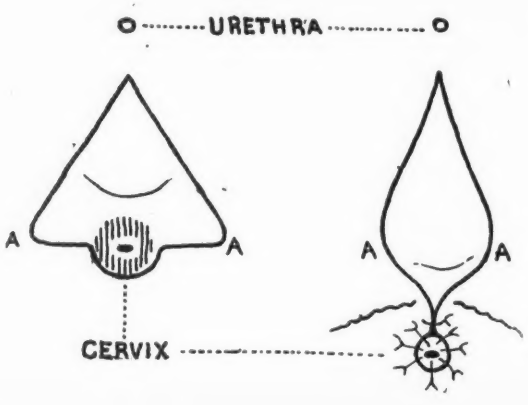
* Read at a Meeting of the North of England Gynæcological Society, held at Sheffield on March 20th, 1914.

6. Cut away a triangular portion of the vaginal wall with its apex just behind the urethral orifice.
7. Cut across the cervix at a suitable level.
8. Stitch the stump of the cervix into the posterior part of the incision, working from behind forward, until it is surrounded by vaginal wall.
9. Close the remainder of the incision from side to side.

By this method the structures removed come away in two separate pieces, the vaginal wall and the cervix. I now suggest that it is more elegant to begin behind the urethra and work from below upwards, and to remove the complete "specimen" in one piece.

The steps of the operation by the new method are as follows:—

1. Dilate the cervix.
2. Pick up four points, one just behind the urethral orifice, one at the junction of the cervix and the posterior vaginal wall, and one on either side of the cervix, about an inch to right and to left of the cervico-vaginal junction.
3. Connect the four points by an incision through the vaginal wall.
4. Begin at the urethral angle of the incision and separate the area of vaginal wall enclosed by the incision from the bladder and the other adjacent structures until it remains attached only to the cervix.
5. Snip round and round the cervix until a sufficient portion of the latter is exposed.
6. Cut through the cervix without splitting it.
7. Stitch the stump of the cervix into the posterior angle of the wound, working from behind forwards, and placing the sutures so that they radiate round the new os uteri.
8. Close the remainder of the incision from side to side.



Even with this improvement the operation demands some time and care. With the most skilled assistance I have not managed to do it, and the perineal repair which follows it in less than half an hour, while at least forty-five minutes is generally required. The results, however, are extremely encouraging.

The specimens shown are examples of the triangular portion of the vaginal wall removed together with the cervix in one piece. They are longer and narrower than they were before removal. The circular muscles of the vaginal wall cause contraction of the portion removed from side to side the moment it is set free from the bladder, para-vaginal, and parametric tissues. Therefore in doing the operation it is necessary to separate the lateral limits of the incision much more widely than inspection of the specimens would suggest. It must be remembered that as the incision is closed from above downwards the stump of the cervix disappears entirely from view, passing upwards and backwards towards the hollow of the sacrum. Therefore catgut only must be used as suture material. Again, interrupted sutures must be used; for mattress or continuous sutures pull the cervix down and forward, while the object of the operation is to push it upward and backward.

I would further repeat that, when this operation is done, less attention to the posterior vaginal wall is required than when older forms of anterior colporrhaphy are used. It is my custom simply to repair the perineum in most cases, leaving the vagina wide enough to admit two fingers. When rectocele is present I do posterior colporrhaphy combined with perineorrhaphy from above downwards by Dr. Donald's well-tried method.

When vaginal work is carried out on these lines it will be found unnecessary to do any abdominal operation for any clinical variety of prolapse. Vaginal hysterectomy, interposition, and other vaginal operations which have been suggested will also be given up. With slight modifications which the circumstances of each case will suggest the operator can deal with cystocele, classical prolapse, inversion of the vagina from above downwards, and rectocele. He will, when possible, avoid amputation of the cervix in young subjects, and will be pleased to find that the operation will often stand the supreme test of subsequent pregnancy and parturition. He will also find that women aged seventy years and more can be relieved of complete prolapses of ancient date without undergoing the risks inseparable from more complicated operations.

SELECT CLINICAL REPORTS.

Aneurism of the Internal Iliac Artery.

By W. E. Fothergill, M.A., B.Sc., M.D., and
Daniel Dougal, M.D.

The subject of this unusual complication was a young unmarried woman, 22 years of age. When seven months pregnant she was sent to St. Mary's Hospital as a case of severe pelvic deformity, and as the external pelvic measurements were extremely small Cæsarean section at full term was chosen as the most suitable obstetric operation. No internal examination was made at this time.

On January 2, 1913, the patient was admitted to Hospital for operation, and this was performed six days later. Recovery was uneventful, the pulse and temperature being normal throughout convalescence.

No vaginal examinations were made by either of us until three weeks after operation, when a hard tumour, the size of a tangerine orange, was felt lying to the left of a small anteverted uterus. There was some pulsation present, which led us to suspect the presence of an aneurism, but as the patient complained of no symptoms we thought that the pulsations might be those of the uterine artery stretched over a cyst in the broad ligament, or possibly transmitted through an old inflammatory exudation. The patient was most anxious to return to her home, and was therefore allowed to do so on condition that she reported herself again at the end of a month. When seen again she still complained of no symptoms and appeared to be in perfectly good health. On vaginal examination the tumour was found to be if anything a little larger than when in hospital, and the pulsations were still present, but not so well marked.

We lost touch with the patient till November 1913, eight months later, when she again came down for examination. Her appearance was now much changed, and she was evidently acutely ill. She stated that she had been very ill for three months, and was gradually getting worse. There had been several fainting attacks, and pain was severe in the left hip extending down into the left leg.

Examination revealed a very alarming state of affairs. An indefinite tumour was felt occupying the lower part of the abdomen and extending as high up as the umbilicus. Per vaginam, the small uterus was found pushed forwards and to the right by a large boggy mass filling the pelvis. The mass was extremely tender, but no pulsation could be made out. The patient was admitted to Hospital

immediately, and an operation arranged for the following day. During the night the pain was more severe, and her condition rapidly became worse. The operation was therefore performed rather earlier, but by this time the patient was pulseless and in a dying condition.

The abdomen was rapidly opened, and found to contain a large quantity of free blood, the pelvis being filled with an indefinite mass to which bowel and omentum were adherent. When examining this more carefully its wall suddenly gave way, and there was a rush of bright arterial blood up from the pelvis. An aneurismal sac had evidently ruptured, but its exact situation could not be made out, and as the patient's condition became critical and forbade any further manipulations, the cavity was tightly packed with gauze, and she was sent back to bed, where she died an hour later.

A complete post mortem examination was not obtained, but the pelvic organs were removed *en bloc* through the abdominal incision, and revealed at once the actual nature of the disease, namely, a ruptured aneurism of the internal iliac artery. The pelvic cavity was occupied by a large sac, the walls of which were composed of successive layers of blood-clot. The upper part of the sac had given way and so allowed the contents to escape into the abdomen. There had evidently been a considerable amount of leakage before operation, but the actual rupture took place whilst the latter was in progress. The pelvic organs were displaced by the sac to a marked degree. The uterus as a whole was pushed forwards against the pubes, but in addition there was a marked left lateriversion, the cervix being pushed over to the right of the middle line. The rectum was lifted up from the sacrum and pushed over to the right side of the pelvis, where it was closely related to the right wall of the sac. Within the sac itself was found a small opening communicating with the main trunk of the internal iliac artery about an inch below its origin and just above its division into anterior and posterior branches. The aneurism therefore belonged to the sacculated type. The pelvic arteries were thin-walled and parchment-like, and showed an excess of fibrous tissue; no definite endarteritis, either simple or specific, could be made out.

With regard to the cause of the aneurism, this has evidently to be looked for in the general condition of the patient's arterial system, possibly in the changes in blood pressure consequent on the pregnant and puerperal states. Traumatism would appear to be ruled out, as the child was not born per vias naturales, and there were therefore no internal manipulations apart from those associated with the Cæsarean operation.

We personally believe that the aneurism was present at the time the patient came under our notice, but unfortunately this is not capable of definite proof. It certainly was present three weeks after the Cæsarean section, and had then attained a large size, and from

that time until the fatal termination, ten months later, its progress was fairly rapid. Supposing that an aneurism were present, what would be the effect upon it of pregnancy, labour and the puerperal state?

With the growth of the pregnant uterus, the uterine artery and consequently its parent trunk, the internal iliac artery, have more and more blood to convey into the pelvis, and therefore there is a gradually increasing strain upon the vessel walls, so that the tendency for any weak spot to give way is increased.

During labour, with the strong and intermittent uterine contractions, there result sudden changes in blood pressure in the pelvic vessels, and these would no doubt prove more injurious to an aneurism or portion of diseased vessel wall than the more gradual changes of the pregnant state.

In the present case, however, the patient had no labour pains, so that this could have had no influence upon her condition. After labour two factors have to be considered. When the uterus contracts firmly down upon the vessels within its walls, there is a sudden change in the pelvic blood pressure, and this would be felt most strongly in the internal iliac arteries, especially at their points of division into anterior and posterior branches. Again, with the sudden diminution in size of the uterus, a considerable amount of external support is withdrawn from the aneurism, which is therefore more liable to give way in the face of increased pressure from within.

In the present case the aneurism was no doubt influenced by both these factors, and probably increased considerably in size during the puerperium, with the result that a point was reached where spontaneous cure became impossible and unfavourable progress of the case inevitable.

Aneurism is a disease of middle life, and it is rare to find it occurring in one so young as the subject of this case. McGraw, in the *Transactions of the American Surgical Association,* has reported 64 cases in young people.

The internal iliac artery is rarely affected with the disease at any age, and there are extremely few cases to be found in the literature of the subject.

The writer in *Keen's System of Surgery* does not mention a single case, and Ericson has only been able to find one out of many thousand aneurisms affecting other vessels.

Little[1], in the *Transactions of the Academy of Medicine in Ireland,* has reported a specimen found in an elderly male dissecting-room subject, but was unable to give any clinical details. The aneurism was of the fusiform type, and involved not only the main trunk but also the commencement of the obliterated hypogastric artery.

More recently, Maclaren [2] has recorded a case occurring after a

confinement in a young woman of 18. Symptoms commenced four days after labour, and in six weeks' time a tumour, the size of a tennis ball, could be felt to the left of the uterus. Maclaren ligatured the main trunk of the internal iliac artery, and the patient made a good recovery. Eighteen months later the tumour, although no smaller, was hard and indurated, but there was still some pulsation to be made out.

Delivery was effected with forceps in this case, and the author considers that a laceration produced by these was probably the exciting cause of the aneurism. This is no doubt a feasible explanation, but if injuries during labour really played such an important part one would expect to find aneurism one of the most frequent complications in midwifery instead of being, as it is, little more than a pathological curiosity.

References.

1. Little, T. G. "Note on a specimen of aneurism of the internal iliac artery." *Transactions of the Academy of Medicine in Ireland.*, vol. 1, 1883.
2. Maclaren, Archibald. "Aneurism of Internal Iliac Artery." *Annals of Surgery*, ii, 269, 1913.

Post Mortem Specimen of Ruptured Tubal Pregnancy: Symptoms Suggesting Irritant Poisoning.

By Wm. Alex. Mackay, M.D., Southport.

The interest in this case lies principally in the history of the illness and of how the specimen came into my possession.

On the evening of March 4th last, I received a telephone request from the secretary of the Grocers' Association to attend, on their behalf, some miles from Southport, the post mortem examination of the body of a lady who had died suddenly shortly after eating some bloater paste, supplied by one of their members. The autopsy was to be made at the instance of the coroner, and in addition to the two medical gentlemen who had attended the case during life, I was to meet a distinguished bacteriologist.

The cause of death was supposed to be poisoning from the bloater paste. Just before the *sectio* the following history was obtained:—

Mrs. E. G., æt. 40, a healthy woman, made a meal of bloater paste, bread and butter, at 4-30 on March 2nd; at 7-30 she took some cold beef; at 10, part of an apple; and went to bed perfectly well. She wakened about 7-50 a.m.—her usual time—apparently well. At 8 a.m. she felt sick, and going to the bathroom, took a dose of Eno's fruit salt. Shortly after 8 she called over the banisters to the servant to prepare breakfast, mentioning the food she required—ham and eggs. She then complained of faintness, went giddy, and had nausea, followed by vomiting. Immediately thereafter she had diarrhœa. The diarrhœa continued with occasional vomiting. I cannot say what quantity of fæcal matter passed—the motions were liquid; apparently, however, her desire to have the bowels moved was so marked as to cause her anxiety that she could not relieve herself decently.

She was seen by her doctor from 10-30—11 a.m. She was then collapsed, pale and bloodless. The pupils were dilated. She was on her right side. The radial pulse was not perceptible, and only a faint impulse was felt in the carotid. Later a certain amount of cyanosis of the lips was noted, but the pallor persisted. She complained of some, but not severe, pain, and was troubled with thirst. In spite of stimulant treatment, including the administration of 20 ounces of normal saline subcutaneously, she died about 1 p.m.

The menstrual history could not be obtained before the body was opened. Later I learned from the husband that she was near a menstrual period, and that the one preceding had been less than usual; indeed he had made some chaffing remark to her of the possibility of pregnancy.

The body was that of a very well-formed and well-nourished woman. Rigor mortis was well marked 45 or 46 hours after death. Pallor was marked.

On opening the abdominal cavity 2 or 3 lbs. of blood and clot were found lying free, and in the right oviduct was a ruptured gestation sac. In the blood-clot an embryo, measuring 18·5 mm. was found. The uterus and tube were removed, and have been preserved. The uterus was nulliparous, and the sound passed in the cavity, after hardening, to 3 in. or thereby. The ruptured right tube was packed gently with cotton wool before being placed in spirit. In its present state it is some 2 in. in length.

The embryo was handed to Dr. Gowland, who is making certain researches. The measurement in the longest diameter is 18.5 mm. and, as will be seen from the photograph handed round, flexion of the neck is still well marked. It is, I take it, at the end of the 6th or the beginning of the 7th week.

Cases of sudden death after rupture of an ectopic pregnancy have unfortunately occurred from time to time. Bland Sutton, in his book on diseases of the Fallopian tube, mentions the case of an English actress, who died suddenly while taking an ice in a café in the Bois de Boulogne. Poisoning was suspected. The death later was proved to be due to a ruptured ectopic pregnancy. He states that many cases of ruptured ectopic pregnancy have only been revealed by dissection instituted by the coroner's order.

The isthmus is supposed to be the most dangerous site for a tubal pregnancy. Our present case is purely tubal—in the centre of the tube at that.

With regard to the possibility of operation had the case been recognised, one must remember that the patient lived some miles from surgical aid, and that 2½ hours passed between the doctor's visit and death—a short period. Had the gravity been recognised at the time of rupture a period of four to five hours should have sufficed to save life. Technical difficulties the operation itself did not exist.

Ovariotomy Followed by Ectopic Pregnancy in Stump.

By Harold Clifford, M.B. (Lond.), F.R.C.S (Edin.), Manchester.

The patient was a married woman, aged 26 years, who had had two living children, and possibly a miscarriage, for which she was curetted in May, 1913. In September, 1913, she had all the signs and symptoms pointing to an ectopic pregnancy on the right side, but on opening the abdomen the tumour was found to be a small cystic tumour of the right ovary, with no sign of pregnancy; this was removed together with the tube. The left appendages were apparently healthy. She made an uninterrupted recovery.

In October she menstruated for five days without pain; she missed the November period, however, but commenced to lose on December 20th, being then three weeks over her usual time; at the time she complained of abdominal pain and faintness. The pain became worse, especially on the left side, and caused her to faint completely on two occasions. About this time I saw her in the out-patient department of St. Mary's Hospitals, Manchester, and found she had a fresh swelling in the pelvis, roughly the size of a three months' pregnancy; she, however, denied the possibility of pregnancy, and as she was going downhill I decided to reopen the abdomen. On doing so I discovered an ectopic pregnancy, apparently in the stump of the previous ovariotomy, but owing to that operation it was difficult absolutely to define its relations to the Fallopian tube and round ligament; there was also a small ovarian cyst on the left side. I removed the cyst together with the uterus and ectopic sac, and the patient made a speedy recovery.

Dr. Shaw, who examined the specimen microscopically for me, found chorionic villi in the sac.

Fig. I.
Showing compressed embryo from Case I.

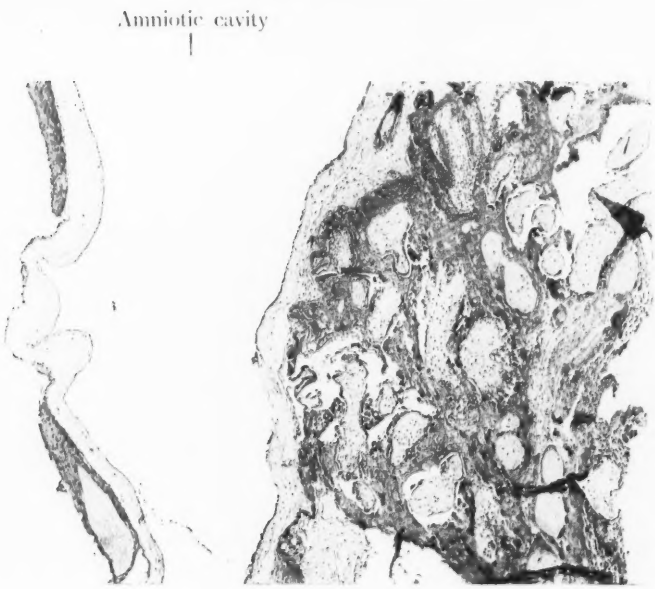


Fig. II.
Section showing compressed amniotic cavity, chorionic villi and decidua. From Case I. (× 30.)

Three Cases in which an Early Twin Ovum was Delivered Together with a Full-Term Child.

W. W. King, M.B., F.R.C.S. (Ed.)

Tutor in Gynæcology and Obstetrics, University of Sheffield.

The following cases are reported, not only on account of their apparent rarity and intrinsic interest, but also because they have an important medico-legal aspect.

The specimens were obtained from three patients at varying intervals after the delivery of full-term children, and they represent the hitherto unrecognised fact that the early death of a twin ovum may simulate the retention of a piece of placenta within the uterus. Two of the specimens were sent to Mr. Miles H. Phillips by a doctor and midwife respectively, and I am much indebted to him for allowing me to make use of them. The last case came under my own observation.

Case I.—This specimen (Fig. 1) was found loosely adherent to the intact membranes of a full-term child delivered by a multipara. The fact that it was passed in this way excluded, of course, any question of negligence, and at the same time suggested its true nature; but it might just as easily have been retained and have given rise to serious trouble.

It is a thin, yellow piece of tissue measuring 12.0 by 0.3 cm. A membrane resembling the amnion is loosely attached to one surface. Microscopic sections taken through the whole tissue show from left to right (Fig. 2) remains of decidua, amnion, amniotic cavity, amnion, degenerated villi, and decidua. The tissue is therefore the compressed embryonic sac of a comparatively early ovum from which the fœtus has been absorbed.

Case II.—The patient from whom this specimen (Fig. 3) was obtained was also a multipara who was delivered of a full-term child. There was no post-partum hæmorrhage. Towards the end of the first week of the puerperium she passed a piece of tissue, which the midwife in attendance brought to Mr. Phillips as an example of "false conception."

The tissue is similar in colour to the last, but smaller and thicker, measuring 8.0 by 0.5 cm. There is a depressed circular area in the centre, to the margins of which a thin membrane is attached which is clearly the remains of the amnion. Microscopical examination shows decidual cells and degenerated villi (Fig. 4). There can be no doubt, therefore, that it is also the remains of an early twin ovum.

Case III.—I obtained this specimen (Fig. 5) from the uterus of a

multipara who had delivered a full-time child ten days previously in the Jessop Hospital for Women. There was no post-partum hæmorrhage, and the puerperium was normal except that the lochia were still red on the tenth day. Finding upon examination that the cervix was patulous and the uterus enlarged, I explored the cavity with my finger, and removed a dark-red piece of tissue, which I mistook on a rather hasty examination for a succenturiate lobe of placenta, whose presence had remained undetected owing to a careless examination of the placenta and membranes by the nurse who delivered the patient. Subsequent examination, however, showed how mistaken I was, for, as can be seen in the photograph (Fig. 5), there is an embryo 0.8 cm. long attached by its umbilical cord to the centre of the mass. The membranes have been opened up to show the embryo more clearly, but in the recent state the amniotic sac was intact, although the liquor amnii had been absorbed. The embryo has been to a large extent destroyed by being mounted in an unsuitable preserving fluid, but when fresh it was easy to see the optic vesicles and limb buds. The death of the embryo probably took place at about the fifth week of intrauterine life.

In each of these cases there was, therefore, the remains of a comparatively early ovum in the uterus, together with a full-term child. This could readily occur in a bicornuate uterus, but would be of little forensic importance seeing that the abnormality could be recognised at any time on physical examination. This condition did not obtain in at least two out of the three cases reported here. In Case I. the ovum was found *adherent* to the membranes of the other child, and must therefore have been fertilized in the same cavity; while in Case III. my digital examination of the uterine cavity excluded any structural abnormality. There is no clue to the condition of the uterus in Case II., which was only seen by a midwife.

I do not propose to discuss the academic question of superfœtation, because to my mind the interest of these specimens lies not in theories as to their origin, but rather in the fact that a blighted twin embryo can closely simulate a succenturiate lobe of placenta. In all of them it has been possible to demonstrate the true nature of the tissue, and hence no question of negligence could have arisen, but if septic infection had taken place this demonstration would have been impossible, and it must be remembered that it is in just such a case of puerperal infection that legal proceedings would be most liable to be undertaken.

The delivery of twins of greatly different stages of a development is, of course, a well recognised phenomenon, but I have only been able to find records of nine cases which are in any sense similar to these which I have described. In four of these cases—reported by Lee, Vines, Thompson, and Schultze and Rolleman—a *full-time child* and *an embryo of ten weeks or under* were delivered at or about

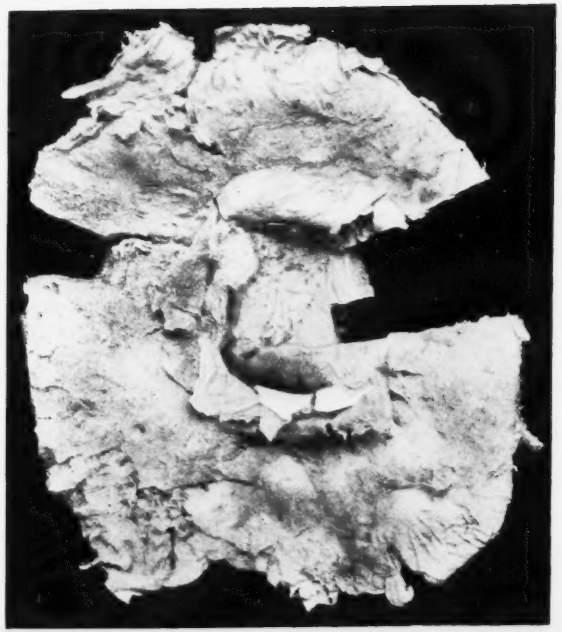


Fig. III.
Placenta and remains of amnion from Case II.

Fig. IV.
Section of placenta of Case II., showing decidua and degenerated chorionic villi. (× 40.)

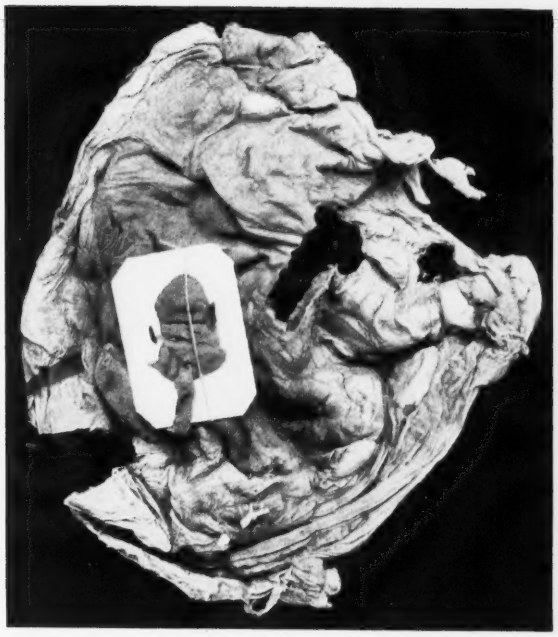


Fig. V.
Amniotic cavity and embryo from Case III.

the same time. The case reported by Vines, in which an eight weeks' ovum was passed 48 hours after a normal full-time labour is the only one of these where any considerable interval elapsed between the two births. The delivery of *premature* children of from five to eight months' and an embryo of ten weeks is recorded by Mann, Strahan, Hartog, and Voigt. In the last case a blighted ovum was passed four days after the birth of a six months' child, and Voigt in reporting it in 1897 states that there are no records of similar cases, but this is incorrect, for Strahan recorded a case in 1883 in which an ovum of two months was delivered two days after the birth of a premature child of between six and seven months. This is the only case in which the author definitely states that the uterus was not bicornuate.

Outhwaite and Eddowes have each reported cases which are clearly examples of uniovular twins, and I exclude these because the placentæ can never be left in the uterus. During the discussion on a specimen of succenturiate placenta shown at the London Obstetrical Society by Champneys, Daly described a case in which a woman died of hæmorrhage caused by a piece of placenta which had been left in the uterus. The patient had been attended by an unqualified man, and, as was proved at the inquest, he had undoubtedly removed the placenta after labour. Just before death a piece of placenta was passed, which Daly saw and regarded as a "supplementary" placenta. The jury accepted this explanation. A modern jury in a civil action might very well refuse—though I do not say with justice—to accept such a plea in the case of a medical man, and unless the suggestion was put forward that the tissue was in fact a blighted twin ovum they would award damages against him.

It is this aspect of the question which I wish to emphasise, because I believe that one result of the Insurance Act will be that actions for negligence will become more and more common, and those who are liable to be called as expert witnesses must be prepared with every legitimate and feasible explanation of cases of apparent neglect.

References.

Lee, *British Medical Journal*, January, 1892, p. 51.
Vines, *British Medical Journal*, March, 1875.
Thompson, *British Medical Journal*, 1898, Vol II, p. 807.
Schultze and Rolleman, (Mentioned by Hartog) *Münch. med. Woch.* 1907. No. 36, p. 1787.
Mann, *British Medical Journal*, 1887, vol. II, p. 493.
Strahan, *Lancet*, 1883, vol. II, p. 813.
Hartog, *Münch. med. Woch.*, 1907, No. 36, p. 1787.
Voigt, *Prager med. Woch.*, 1897, No. 321.
Outhwaite, *Trans. Obst. Soc. of London*, 1882, vol. 24, p. 3.
Eddowes, *British Medical Journal*, 1897, vol. II, p. 597.
Daly, *Trans. Obst. Soc. of London*, 1883, vol. 25, p. 214.

Description of a Specimen of Allantoidoangiopagous Twins.

By Daniel Dougal, M.D.,

Hon. Assistant Surgeon, Manchester Northern Hospital; Pathologist, St. Mary's Hospitals.

Allantoido-angiopagous twins are uniovular fœtuses which communicate with one another by means of the vessels of their umbilical cords near to or within the single placenta. As a rule, one of them is fairly normal in appearance, but in the other the deformity is generally of an extreme type. The fœtuses are always of the same sex, and have sometimes been called placental parasites, but Ballantyne[1] considers this to be a misnomer, as one twin is really a parasite on the other and not on the placenta.

There are three chief groups of this type of monstrosity, the paracephalic, the acephalic, and the amorphous respectively.

The paracephalic type is distinguished by the presence of a head, the other parts of the body showing varying degrees of deformity.

In the acephalic monster there is, of course, no head, whilst the amorphous form lacks the external appearances of a fœtus, and may consist of little more than an oval skin-covered mass.

The specimens which I have dissected, and which form the subject of this communication, present many interesting features, the larger twin being anencephalic, whilst the former one belongs to the amorphous group already mentioned.

The larger fœtus measures twelve inches in length and weighs 22 ounces. It is of the female sex, and presents the typical appearances of an anencephalic monster.

The umbilical cord measures seven inches in length and contains two vessels, there being only one umbilical artery present.

This is an interesting abnormality in itself, and on making a dissection it is found that this single artery is a continuation of the abdominal aorta, and gives off within the abdomen the right common iliac artery and close to its point of entrance into the cord, the superior vesical artery.

The common iliac on the left side arises directly from the aorta, and much higher up.

The internal organs of this fœtus are of normal appearance with the exception of the heart, which is somewhat hypertrophied, due, no doubt, to the increased work thrown upon it by the parasitic twin.

The smaller fœtus resembles a kidney in appearance, and has a portion of umbilical cord attached to it very much in the same position as the ureter in the case of that organ.

Plate I.

Showing relative size of Twins.

a. Larger, anencephalic fœtus.
b. Smaller, amorphous fœtus.

It measures $3\frac{1}{2}$ inches in length, $1\frac{3}{4}$ inches antero-posteriorly, and 1 inch laterally, and weighs about an ounce and a half.

There are no sexual characteristics present, but the sex is presumably the same as that of the other twin.

External Appearances.—The surface of the fœtus is of a deep brownish-red colour, and thrown into numerous wrinkles.

Fine hairs are present over the whole surface, but are more numerous near the upper end over the part corresponding to the scalp.

There is no trace externally of limbs or limb buds.

Near the upper end of the fœtus and on its anterior surface are four fleshy processes enclosing a deep fissure. These represent the fronto-nasal, maxillary, and mandibular processes, and surround the imperfectly-formed mouth of the fœtus.

About the same level and on each lateral surface is a small pit, which may represent the external auditory meatus.

Hard, irregular masses are felt through the skin, and a radiogram of the fœtus shows these to consist of masses of cartilage enclosing smaller areas of bone formation.

Osseous System.—A study of the radiogram and of a mesial section of the fœtus shows that the bones of the skull and vertebral column are fairly well developed, and enclose a large cavity containing the central nervous system.

Within the fronto-nasal process the cartilage is hollowed out into numerous cavities, which evidently represent the fronto-ethmoidal cells.

At least four ribs can be made out on each side and behind, and on the outer surface of these several small, irregular portions of bone probably correspond to an incompletely-developed shoulder girdle.

The lower end of the vertebral column is bent forwards to form an angle of about 80° with the upper part, and is composed entirely of cartilage.

The pelvic bones are represented by a bony-cartilaginous mass, which is found on section to be composed of four separate pieces. In only one of these has ossification taken place, and this probably represents the ilium, the centre for which appears about the end of the second month of intra-uterine life.

The other internal structures of the fœtus were studied in a mesial section.

Opposite to the entrance of the vessels of the umbilical cord is a large cavity evidently forming the coelom, and containing a single organ, the heart.

There are no abdominal organs to be found.

Two vessels enter from the cord, the vein running up to the heart, the artery coursing downwards in association with a structure representing the allantois, and immediately behind the mass of cartilage lying at the pelvic end of the fœtus.

It was impossible to make a complete dissection of the vascular system, but a comparatively large vessel was found running upwards in front of the vertebral column, and evidently forming a primitive aorta.

The heart is a fairly large organ, with rather thin walls, and containing a single chamber, lying within the coelom, the diaphragm being totally undeveloped. Immediately above the heart is the cavity of the pharynx, whilst behind the vertebral column is in close apposition with it. The circulation, such as it is, probably consists of a simple vascular circle connected with the heart, but the latter organ has evidently had little functional value, to judge from its thin-walled and dilated appearances, and the stronger heart of the other twin evidently overpowered it and usurped its functions.

The central nervous system is present, the spinal cord being especially well-developed, but the faulty preservation of this tissue prevented any detailed examination being made.

The common placenta measures $6\frac{1}{2}$ inches in diameter and weighs 12 ounces.

The cord of the larger fœtus has a velamentous insertion, and shortly after joining the placenta proper the umbilical artery and vein each give off a small branch, and these, coming together, course outwards on the surface of the chorion in similar fashion to the vessels which supply a succenturiate placenta.

There is no doubt that these vessels represent the vascular attachment of the parasitic fœtus, which, therefore, does not get a supply of freshly-oxygenated blood, but only obtains it second hand from its more powerful partner.

Opinion differs widely as to the origin and mode of development of this type of monstrosity. The most probable explanation is that one fœtus obtains a larger share of or an earlier attachment to the chorionic vessels, and as a result starves out its less fortunate companion.

The latter, if left to its own resources, would probably die, but it appears as if the other twin, having gravely interfered with the nutrition of its fellow, now proceeds to carry on the latter's circulation in a reverse direction and with imperfectly oxygenated blood, with the result that growth continues, but along abnormal lines.

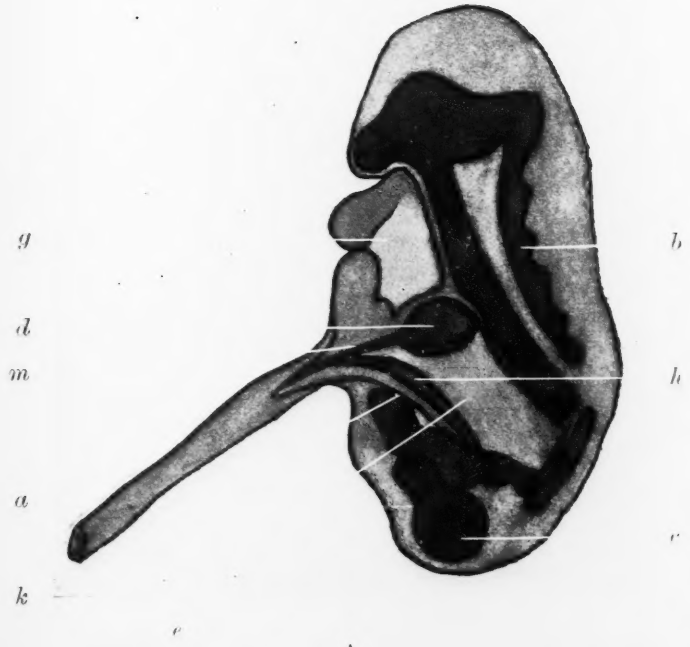
A second view holds that the larger fœtus plays a beneficent rôle from the very beginning, and that the development of the smaller one is so imperfect that ante-natal existence would be absolutely impossible but for the help received from the other's circulatory system.

For the specimens above described I am indebted to Dr. W. K. Walls, to whom they were sent by Dr. Moir, of Darwen.

Reference.

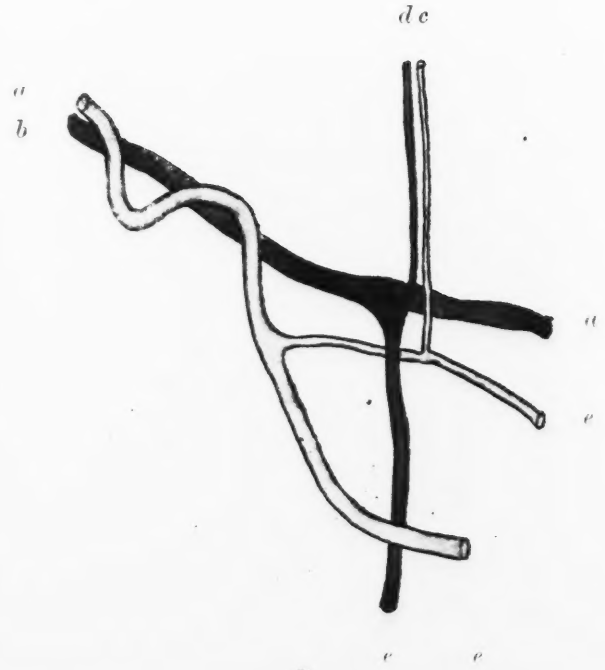
(1) Ballantyne, J. W., Manual of Antenatal Pathology and Hygiene. Vol. 1. The Embryo. p. 623.

Plate III.



A

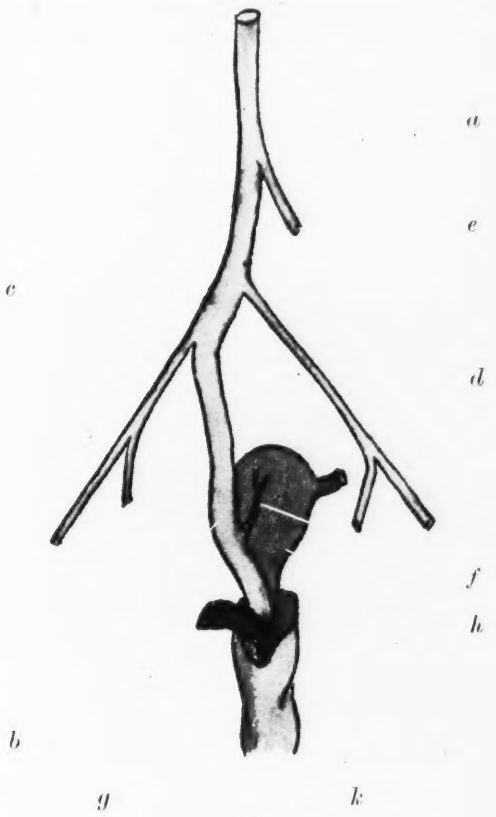
A. Mesial Section of Amorphous Fœtus. (Semi-diagrammatic).
a. Umbilical cord. *b*. Vertebral column. *c*. Mass of bone and cartilage representing pelvic bones.
d. Heart. *e*. Coelom. *g*. Pharynx. *h*. Umbilical artery. *k*. Allantois. *m*. Umbilical vein.



B

B. Diagram of Vascular connections in the Placenta.
a. Umbilical artery and *b*. umbilical vein of larger twin. *c*, *d*. Artery and vein running to smaller twin. *e*. Placental vessels.

Plate III. (continued).



C

C. Diagram of Single Umbilical Artery in larger twin.

a. Abdominal aorta. *b*. Umbilical artery. *c*. Right common iliac artery. *d*. Left common iliac artery. *e*. Inferior mesenteric artery. *f*. Superior vesical artery. *g*. Umbilical vein. *h*. Bladder. *k*. Umbilical cord.

Case of Double Ovarian Cyst.

By Miles H. Phillips, F.R.C.S., Sheffield.

It would seem very improbable that an ovarian tumour of about the size of a full-time fœtal head could be overlooked during a laparotomy. The oversight was committed under the following circumstances.

Mrs. E. H., a multipara of 68 years, had thought that her abdomen was unduly enlarged for some eighteen years. It had certainly been rapidly increasing in size for three years. There had been slight uterine hæmorrhage for two months. She was a thin, feeble woman, with greatly thickened arteries and extensive chronic bronchitis. The abdomen was enormously enlarged by a cystic swelling, the greatest diameter being 47 inches. She was a very bad subject for operation, and it was very desirable that an uncomplicated ovarian cyst should be found.

On February 13th, 1914, the abdomen was opened. There was no free fluid and no adhesions. The cyst was tapped and 26 pints of fluid easily removed from three large loculi. It was then seen that the cyst extended deeply into the left broad ligament and the pelvic meso-colon. It had also dragged the uterus up into the abdomen. It was necessary to put the patient into the Trendelenberg position to facilitate the pelvic dissection. This was carried out satisfactorily, and the cyst was removed with the body of the uterus. No time was then lost in closing the abdomen, and the condition of the right uterine appendages was not noted.

There were no abdominal complications, but a sharp attack of bronchitis caused considerable anxiety for several days. Thereafter progress was steady until nearly three weeks after the operation, when the patient was sitting up. She then complained of slight abdominal pain. This recurred on the following three days, and became more severe. On removing the binder a swelling was plainly visible, bulging the thin abdominal wall to the right of the umbilicus. It was slightly tender and of the shape of an ovid disc, with irregularly-rounded edges. It was freely movable, and could be readily pushed under the left costal margin. There was no rise of temperature and no intestinal symptoms.

In considering its nature it seemed impossible that a tumour of the right ovary of the size of this swelling could have been overlooked during the first operation. The absence of any rise of temperature and of intestinal symptoms, together with the free mobility, were taken to negative it being due to a swab or other foreign body.

Both kidneys could be palpated in their normal positions, so a provisional diagnosis of torsion of a dislocated spleen was made.

The patient was very unwilling to face another operation, but a more severe attack of pain a few days later led her to give consent. It was then found that the tumour was an ovarian cyst (measuring 5 by 4 by 4 inches), with a thin pedicle six inches in length and many times twisted. A thick band of great omentum was adherent to the cyst, and it was evident that this band had tended to pull the tumour upwards and to the left. This, combined with the Trendelenberg position at the original operation, had led to the tumour being withdrawn from the field of operation. Anxiety to conclude the first operation rapidly led to neglect of the routine examination of the other ovary. The patient made a good recovery.

The Contraction Ring as a Cause of Dystocia.

By V. B. Green Armytage, M.D., I.M.S.,

Resident Surgeon, Eden Hospital, Calcutta.

This case which I venture to report, appears to be of interest from the fact that the contraction ring was not the result of obstruction but the cause of it. This is, I think, the rarer of the two conditions, for out of 7 cases I have had to deal with 6 have been associated with some form of obstruction to delivery in the bony pelvis.

Mrs. C., æt. 28, primigravida, full-term, gave a history of the membranes having ruptured, without any pains coming on, three days previously. When seen she was having slight pains; the pulse was 100, the temperature 99°F.; the abdomen was of normal size; the head was not engaged in the brim, nor was it enlarged. The position of the fœtus was L.O.A. Heart sounds healthy. Pelvic measurements, internal and external, normal. The os was the size of three fingers, the cervix taken up, the head quite mobile and high up above the brim.

She was ordered morphia gr. ½, and during the next 16 hours the pains increased in power somewhat, though the local conditions remained exactly as they were before.

Suddenly, 20 hours after I first saw her, I was asked to see her again as she had shown signs of distress; the pains were coming every two minutes; the pulse was 140, and the temperature 101. On examining the abdomen there was no change in the condition whatever, except that there was now a definite sulcus to be felt across the uterus about four fingers' breadth above the symphysis.

Suspecting contraction ring as the cause of the trouble, the patient was deeply anæsthetized. Then, placing the whole hand in the vagina, I was able to dilate the cervix to full size, and then passing my hand with difficulty along and above the head, I was able to feel a tight ring constricting the neck, which corresponded with the sulcus felt on the belly wall. The fœtal heart sounds were now slow and feeble. High forceps were tried twice, but each time they failed, from slipping; so the head was perforated and brain matter washed out; and then, using first the cranioclast and later Auvard's combined instrument, I tried hard, but failed completely to deliver, though steady prolonged traction was used with the hope of relaxing the ring. The instruments were then taken off. I was just in the process of cutting through the clavicles, which was a very difficult operation from the height of the head and the closeness of the ring, when the patient quite suddenly expired from heart failure.

The fœtus and the uterus were at once removed from the corpse. There was no rupture of the uterus. As a demonstration of the contraction ring the specimen is a perfect one; the ring was tightly round the neck, and I do not think anything short of embryulcia would have been successful, as post mortem I attempted to drag the child past after severing the clavicles and failed. The lower uterine segment was not thinner than normal.

The points of interest in this case are :—

1. The early rupture of the membranes with very delayed pains.
2. The head remaining mobile above the brim to the very last.
3. The contraction ring being round the neck, preventing delivery of the shoulders.

The problem of treatment in these cases, which are seen late and are potentially septic, is a very difficult one: and from an ethical point of view, those where the the contraction ring is the cause and not the result of the obstruction are still more so :—

1. From the point of view of Cæsarean section with the ring round the child's neck, the "life" of the child must be deemed "bad."

2. Early rupture of the membranes, plus delay, plus probable multiple examinations, suggests septic complications after Cæsarean section, which in these cases means incising right into the lower uterine segment.

3. Even though the child's life is negligible, or if it is dead, we see in this case the great difficulties which may be met with.

Others have reported cases where, although the ring was the only cause of dystocia, and the child was dead, they were able to perform embryotomy successfully. But I am of the opinion that in another such case laparotomy with or without removal of the uterus with the fœtus intact would be the better operation, for the risk of death from rupture of the uterus, shock and sepsis, when working from the vaginal route, are very great.

In two cases where Cæsarean section was combined with hysterectomy, for contraction ring associated with pelvic deformity, I lost one from general peritonitis.

Mammary Murmurs of Lactation.

By E. Marshall Cowell, M.D., F.R.C.S.

In the routine examination of the heart in the lying-in woman there is occasionally to be heard a curious basal bruit, puzzling to the observer encountering the phenomenon for the first time. This murmur is heard over the pulmonary or aortic area, sometimes it is bilateral. The bruit is quite localized and disappears on pressure. In quality the sound is peculiarly harsh and superficial, always systolic in time. In the present series of 400 cases examined in the puerperium the murmur was found 10 times, appearing on the third or fourth day and not usually to be found after the eighth.

The exact spot at which the murmur is produced, is found on close examination to coincide with the presence of a large mammary branch of the internal mammary artery crossing over the second or third rib. In one thin, small-breasted multipara the dilated vessel was actually to be seen making a loop round the lower border of the third rib. The additional factor in the production of the bruit is the low arterial tension so often found in healthy women in the early days of the puerperium. Unfortunately it was only measured in two cases, and then found to be 100 and 108 mm. HgG.

This physical sign does not seem to have attracted notice hitherto, since I can find no reference in the literature to any murmur of the sort. Clinically, this bruit is interesting, since, although the explanation is so simple, I have known it mistaken for a " hæmic " murmur, an aortic bruit or even a sound of pericardial origin.

In conclusion, I wish to thank Dr. Herbert Spencer and the staff of Queen Charlotte's Hospital for permission to publish results of observations under their care at University College Hospital and Queen Charlotte's respectively.

Table of Cases.

No.	Age.	Pregnancy.	Day.	Position.	Artery.	Remarks.
1	26	first	3	2R. rib	felt	Breasts normal.
2	28	third	4	2R. 3L.	„	Small breasts. Art. visible.
3	22	first	3	2L.	not felt	Breasts normal.
4	24	second	1	2R. 3L.	felt	„ „
5	26	first	4—6	2R.	not felt	Breasts large & pendulous. B.P. 108. Rheumatic mitral disease also present.
6	19	„	4—6	2R.	„ „	B.P. 100mm. Hg.
7	20	„	3—7	2L.	„ „	P. anæmic with hæmic m.
8	36	third	7	2R.	felt	Not altered by feeding.
9	35	second	4	2R. 3L.	„	
10	22	„	5	2L.	„	

REVIEWS OF CURRENT LITERATURE.

UNDER CHARGE OF EARDLEY HOLLAND, M.D.

Gynækologische Rundschau.

viii, *Heft* 4, 1914.

1. *The effect of the suprarenals on calcification of bone. J. NOVAK.
2. Sacro coccygeal teratomata. E. VOGT.
3. The effects on the ovaries of X-rays. H. EDELBERG.
4. The use of "Uteramin Zyma." S. KALDORI.
5. Pathology of uterine carcinoma. F. COHN.
6. Infantile skin diseases. C. LEINER.

The effect of the suprarenals on calcification of bone. The author removed the suprarenals from a number of rats. After an interval he examined the skeletons to determine if any changes had occurred in the calcification of the bones. The results were absolutely negative, disproving Bossi's statement that symptoms of osteomalacia occur.

viii, *Ht.* 5, 1914.

1. An improved aspirator. L. KNAPP.
2. Medicinal uterine tonics. E. EKSTEIN.
3. Experiences with noviform. NEBRESKY.
4. Noviform. F. GROAK.
5. Critical review of uterine sarcoma. M. KUNKREUTHER.

viii, *Ht.* 6, 1914.

1 *A rare malformation of the genital tract. O. VON FRANQUE.
2. Combined pregnancy. F. PRIMSAR.
3. Physiology and mechanism of labour. A. LABHARDT.
4. Obstetrical operations. F. JAEGER.

A rare malformation of the genital tract. The author describes the case of a 17-year-old girl, who was operated upon because of severe abdominal pain. The hymen was intact, the vagina rudimentary and no uterus could be felt. The pelvis was found to be empty. Lying in a peritoneal recess on the outer side of the left iliopsoas was a mass composed of ovaries, tubes and rudimentary uterus. He considers the anomaly to be due to non-union of the Müllerian ducts. The development of the genital tract is referred to in considerable detail.

R. M. ALLAN.

Precocious development of the external genitals due to hypernephroma of the adrenal cortex.

HENRY D. JUMP, M.D., HENRY BEATES, Jr., M.D., and W. WAYNE BABCOCK, M.D. (*The American Journal of the Medical Sciences*, April, 1914). The authors describe in detail one case, and then give a brief *résumé* of the literature on the subject. Their case is that of a girl, one of twins. The family history was unimportant. The child was apparently normal till one year old, when rapid growth commenced accompanied by the appearances of hair on the pubes, axillæ, legs and trunk. The voice was noticed to be deep when the child commenced to talk. At six years old the voice was bass, and the larynx and vocal cords, on laryngoscopy, resembled those

of an adult man. When seven years old the skin of the face became coarse and red from acne, and a beard and moustache appeared. There was no pigmentation of the skin. Menstruation had not commenced, the nipples were of masculine type. No abdominal tumour was noticed, although frequently sought for, until the child was seven years and three months old, when a mass was observed in the right hypochondrium, which grew rapidly and was painless. The height of the child at this age was 4ft. 5in., her weight 6st. 6lbs. Her physical and mental development were phenomenal. The configuration of the child and the distribution of hair were of the male type. The clitoris was greatly hypertrophied. The tumour was removed, when the child was seven years and five months old, with great difficulty; the child, however, died three hours later. The growth involved the right kidney, and was found on section to be a hypernephroma of the right adrenal. The uterus, tubes and ovaries were found at operation to be infantile in type. A limited post-mortem revealed no metastases. The authors have collected from the literature seventeen other cases—fourteen girls and four boys. An overgrowth of hair and bodily over-growth were noticed in all cases. Menstruation had occurred in one case only. Pigmentation of the skin was present in many. The development of male characteristics in girls and an intensification thereof in boys was noticed in the majority of the cases. All the cases died before the age of sixteen years. On this account the authors advise operation as soon as the diagnosis is made, although they state that these cases stand operation extremely badly. The authors offer three hypotheses to explain the abnormal development. (1) That tumours of adrenal tissue affect the development of the tissues arising from the intermediate cell mass, *i.e.*, the ovaries and testes, and that the hyperplasia of the sex glands causes the excessive development. This, however, appears to be negatived by the absence of changes in these organs in seventeen out of the eighteen cases. (2) That the tumour has increased the internal secretion of the adrenal cortex, and that this secretion itself influences growth. (3) That the balance, produced by the linking up of the secretion of the ductless glands, not being maintained, owing to the loss of one suprarenal, may cause these changes. In some of the cases thyroid hypertrophy was present. The authors mention in conclusion other causes of precocious development. (1) True sexual maturity in girls due to tumours of the ovaries. (2) True sexual maturity in boys with tumours of the testes, pineal gland or adrenal body. (3) Dystrophia adiposo-genitalis in which there is over-growth of the fat with infantile mental and sexual development.

GORDON LEY.

Further experience in treating carcinoma by mesothorium.

DÖDERLEIN and SEUFFERT (*Münch. med. Wochensch*, No. 5, February 1914), report on their work for the past year, but as five years are necessary before a permanent cure can be assured, they can give no definite results. There was a great increase in the number of advanced cases of cancer during the year, due to the knowledge by the laity of this new treatment. Of 153 observed cases of uterine carcinoma, 24 died; four in hospital and the remaining 20 soon after being discharged. In no case could death be attributed to the treatment.

In 31 cases the mesothorium treatment caused all signs of the carcinoma to disappear. In some cases there was found in place of the former carcinomatous excavation, a healthy mucous membrane covering

the cervix. The tissues were no longer friable, so that the strongest pressure with the fingers produced no bleeding. In some cases the uterus was small and movable and slight induration occurred due to the inflammatory reaction from the treatment. Of 31 cases 19 were suitable for operation; the other 12 being advanced cases, where the good results were unexpected. The authors quote in detail the history of four cases; also a case of cancer of the cervix complicating pregnancy at the third month. The uterus was emptied, and eight days later the mesothorium treatment was begun, the patient receiving three applications in all. The carcinoma disappeared quickly; the cervix seemed normal, differing in no respect from a healthy condition. This case is of special interest, because of the unfavourable prognosis in such cases. D. and S. describe cases of recurrent cancer after operation. The general impression is that such cases are more unfavourable for this treatment than primary ones; still two cases are reported where the recurrent carcinomatous nodules disappeared completely. Eleven cases of cancer of the rectum are quoted, in two cases the disease was arrested, in one after six, in the other after four, sittings.

In two cases of mammary carcinoma the nodules disappeared. In a case which had been operated on three years previously, the recurrent growth on the cicatrix was in no way benefited by the treatment. Their experience of the treatment of mammary cancer is that it requires to be long continued, and that in recurrent cases the results are not satisfactory.

The treatment is painless, but sometimes a febrile reaction sets in lasting some weeks, which is regarded as a favourable omen. In order that absorption should be limited they recommend preparatory treatment in the way of thoroughly scraping away the diseased tissues, and hæmorrhage is to be checked by the application of a neutral solution of iron.

As untoward results they mention intestinal symptoms, in the form of painful dæfecation, and prolonged tenesmus. In two cases a distinct stricture of the rectum was noted. In four cases fistula occurred, but the disease was rather advanced before treatment commenced. Hæmorrhage from erosion of blood vessels has never been observed. On the contrary, prompt arrest of hæmorrhage frequently follows this treatment. In describing the technique it is pointed out that each case must be considered individually, and treated according to the progress of the disease. Comparatively mild doses are employed, from 100 to 200 mgs. On the whole the report of this treatment is favourable, but further work must be done before permanent cures can be claimed.

J. A. Kynoch.

On the ætiology and bacteriology of leucorrhœa.

Arthur H. Curtis (*Surgery, Gynæcology and Obstetrics*. March, 1914). This article is a more or less detailed bacteriological account of the flora of leucorrhoeal discharges, and is not, therefore, one which lends itself to abstraction.

The author finds that the greater number of bacteria in these discharges can only be grown under anærobic conditions and he thinks that these organisms play an important part in the production and maintenance of leucorrhœa. It is noteworthy that cultures made from the region of the vulva, in contrast to those taken from the upper part of the vagina, contain more anærobes, notably B. coli, staphylococci and pseudo-diphtheria bacilli; consequently high vaginal cultures from patients who have been

subjected to frequent douching show contamination with these bacteria. Cultures from the uterine cavity were found to be sterile. The author also states, but without submitting his evidence, that in unmarried women gonorrhœal infection precedes the development of chronic leucorrhœal discharges in the great majority of cases.

W. W .K.

Hunterian lecture on the physiology and pathology of uterine hæmorrhage.

H. B. WHITEHOUSE (*Lancet*, March 28th and April 4th, 1914). The first lecture deals chiefly with Physiological Uterine Hæmorrhage, and is divided up under the following headings :

(1) The relation of menstruation to pro-œstrum.

(2) The menstrual clot.

(3) Menstrual discharge in the human female.

(4) Evidence of a specific thrombolysin in the uterine secretion.

(5) The influence of thrombo-kinase in promoting the clotting of menstrual blood.

(6) The histological characters of the menstruating endometrium.

(7) Factors in the production and cessation of the menstrual hæmorrhage.

It includes the following tables :

I. Showing results of experiments undertaken to ascertain presence of anti-thrombin in menstrual blood and the endometrium.

II. Showing incidence of thrombi in uterine and vaginal blood.

III. To show the thrombolytic power of menstrual blood.

IV. To show thrombolytic power in the fluid obtained by centrifugalising menstrual blood.

V. To show thrombolytic power in menstrual fluid after thrombolysis has occurred.

VI. To show fibrinolytic power of menstrual blood upon ox fibrin.

VII. To show the effect of mucin, calcium salts, lactic and butyric acids upon the coagulation of blood.

VIII. To show the effect of extracts of sheep's endometrium and ovary containing corpus luteum upon the uterus and ovaries of rabbits.

The second lecture deals with Pathological Uterine hæmorrhage, and is divided as follows :

(1) Factors in the production of excessive bleeding.

(2) The endometrium in relation to pathological uterine hæmorrhage.

(3) The importance of investigation of the menstrual discharge.

(4) Influence of the endocrinous glands upon uterine hæmorrhage.

(5) Hæmorrhage at puberty and the menopause.

(6) Hæmorrhage in relation with organic disease of the internal sexual organs.

(*a*) *Uterus*—Displacements—Fibrosis and Chronic Metritis—Endometritis—Hypertrophy of the Endometrium.

(*b*) *Ovary*—Ovarian cystomata—Displacements of the Ovary—Hypertrophy of the Ovary.

(*c*) *Pelvic inflammation.*

(7) Principles of treatment.

(8) Indication for treatment.

With one table :

IX. (*a*) Showing incidence of excessive uterine hæmorrhage in relation with organic disease of the internal sexual organs.

(*b*) Summary of Table IX. (*a*).

(*c*) Contrasting the incidence of menorrhagia in uterine, ovarian and inflammatory disease.

J. M. W.

Adenomyoma of the recto-vaginal septum.

THOS. S. CULLEN (*Journal Amer. Med. Ass.*, Vol. 62, page 835). In the first case the uterus was removed for fibroids with the appendages, and two small myomata were shelled out of the left side of the pelvic floor, from between the rectum and the vagina. On examination typical myomatus tissue was found with islands of typical uterine mucosa. In the second case the adenomyoma was situated in the left broad ligament, and intimately blended with the uterus. The patient was 37 years of age, and two years previously had had a fibroid uterus and enlarged ovaries removed. The patient came under observation for almost continuous bleeding from the cervix. There was thickening behind the cervix and induration in both broad ligaments. On opening the abdomen the rectum was found to be adherent to the bladder, and the left broad ligament was filled with a somewhat cystic mass; this was shelled out from its attachment to the pelvic wall and was found to contain chocolate-coloured fluid. On examination the wall consisted of non-striped muscle fibres, and quantities of uterine glands embedded in their characteristic stroma.

C. N. L.

Prolapse of ovary: its rational management.

CHAUNCEY D. PALMER, M.D. (*American Journal of the Medical Sciences*, April, 1914). The author first tabulates the symptoms of the condition, after stating that the prolapsed ovary is more frequently the left. The differential diagnosis is next briefly discussed. The author advocates treatment by diet, by regulating the bowels and by improving the general nervous tone. He points out the sins of tight-lacing as increasing intra-abdominal tension. He advocates postural treatment, the genu-pectoral position night and morning for twenty minutes with avoidance of lying on the back. He considers that boro-glyceride tampons may be of value applied twice weekly. Of surgical methods, he advocates conservation of the ovary when possible with suspension of the infundibulo-pelvic ligament; if serious structural changes are present he advises oophorectomy. For the left-sided pelvic pain he advocates Faradism, and in long-standing cases the galvanic current, the positive pole placed in the posterior vaginal fornix, the negative pole on the lower abdomen.

GORDON LEY.

The need for research in ante-natal pathology.

AMAND ROUTH (*British Medical Journal*, April 25th, 1914), in a paper read before the Obstetrical and Gynæcological Section of the Royal Society of Medicine, having mentioned the estimated proportion of still-births to abortions, continues with a review of their causes and ideas as to their investigation.

Distinguishing between embryonic and fœtal pathology, he divides the causes into

(i.) The toxæmias of pregnancy.

(ii.) General diseases of the mother.

(iii.) Syphilis.

(*a*) Evidences of syphilis in the still-born fœtus.
(*b*) Placental syphilis.
(*c*) Infection of the fœtus.
(*d*) The effect of syphilis in causing abortions and still births.
(*e*) Colles's law.

(iv.) Ante-natal tuberculosis.

He concludes by discussing methods of providing facilities for research.

J. M. W.

The total non-protein nitrogen of the blood in pregnancy and eclampsia.

CLIFFORD B. FARR, M.D., and PHILIP F. WILLIAMS, M.D. (*The American Journal of the Medical Sciences*, April, 1914). This paper is based on the findings in twenty pregnant women, two-thirds of whom showed toxic phenomena. The technique was that of Folin. The amount of blood required being five to ten c.c. In seven cases of normal pregnancy, the total non-protein nitrogen ranged from 21 to 31 mg. per 100 c.c., and the urea ammonia fraction from 7 to 15 mg. per 100 c.c. In three cases of renal insufficiency, in which pregnancy had to be terminated on account of increasing severity of symptoms, the total non-protein nitrogen ranged from 34 to 52 mg. per 100 c.c., and the urea ammonia fraction from 11 to 30 mg. per 100 c.c. In seven cases with convulsions the total non-protein nitrogen ranged from 38 to 72 mg. per 100 c.c. in six of the cases, the two extremes being fatal cases. In the seventh case, with one post-partum fit only, the non-protein nitrogen was 26 mg. per 100 c.c. In three cases one of chorea, one of retinitis with high blood pressure, and one of uncontrollable vomiting, the total non-protein nitrogen ranged from 21 to 24 mg. per 100 c.c., and the urea ammonia fraction from 8 to 12 mg. In fifteen of the twenty cases the renal efficiency was tested by the phthalein method. It showed a correspondence to the nitrogen retention. The writers conclude (1) that the total non-protein nitrogen during normal pregnancy does not exceed 30 mg. per 100 c.c. (2) That it is always slightly or even considerably raised in toxic conditions and in renal insufficiency. (3) That the increase bears no relation to the severity of the case. (4) That the phthalein elimination is, judging from this series, of no diagnostic value. (5) That the blood pressure, the condition of the urine and the clinical picture are all and severally of more value than either of the methods detailed.

GORDON LEY.

Two cases of pregnancy in uterus subseptus.

R. C. BUIST (*British Medical Journal*, April 25th, 1914) reports on two cases with this abnormality of the uterus, which terminated in the early months of pregnancy; both cases had previously had normal labours. The conclusions he comes to are that it was only coincidence that in these two cases the pregnancy terminated early; the chief disturbances attributed to the condition being

(1) Menstruation occurring during pregnancy.

(2) The formation of decidua in the second cavity, and its later expulsion giving rise to an unusual form of hæmorrhage intra-partum or post-partum.

(3) The unequal development of the uterine walls providing irregular contractions during labour or post-partum, so causing delay in delivery or post-partum hæmorrhage.

(4) Rupture of the irregularly developed uterus.

J. M. W.

Pituitrin in labour.

P. A. HENDLY (*British Medical Journal*, April 25th, 1914) gives a record of 60 cases in which he has used pituitrin, mostly given in the early stages of labour.

He states that ordinary caution should be used in its administration, especially when there is any obstruction, but that it is a very valuable aid towards shortening the long and often exhausting first stage of labour, especially in those cases in which the membranes rupture early.

He discusses its value in cases in which shock and collapse follow prolonged chloroform anæsthesia, also that post partum retention of urine is avoided, and concludes by giving the dosage he uses. J .M. W.

Further experience of posterior cervical Cæsarean section.

POLANO (*Münch. Med. Wochensch*, No. 15, Ap. 1914) gives a short historical account of previous suggestions for the carrying out of this method. He then describes his own method of making a long vertical incision through the lowest part of the posterior wall of the lower uterine segment. He refers to its advantage in permitting free escape of lochial discharge, when the operation is performed before or during the early stages of labour. Other authorities are quoted who favour this method. Truzzi reports 12 successful cases and he emphasises the simplicity of the technique, the less risk of peritonitis; and the better involution during the puerperium. P. reports 7 cases in detail. Of those, 2 were severe cases of eclampsia, 1 a repeated posterior cervical operation, and in 3 the anterior wall was firmly adherent as a consequence of a previous suprapubic Cæsarean section. One of the eclampsia cases was fatal. In the case where the operation was repeated the cicatrix was perfect. He mentions 3 objections put forward to his method: 1st, Danger of asphyxia to the child, resulting from interference with the circulation as a consequence of compression of the uterus against the symphysis-pubis; 2nd, hæmorrhage; 3rd, risks from the long abdominal incision. The first objection is met with by the fact that Truzzi describes 22 cases where there was not the slightest sign of asphyxia.

With regard to the danger of hæmorrhage as reported by Rühle, P. believes that on account of the incision being made far away from the seat of the placental attachment, there is much less risk of hæmorrhage, and he thinks that the untoward effect in Rühle's case was due to the extreme pendulous condition of the abdomen. In discussing the objection to the large abdominal incision P. states that he uses a much smaller incision now than previously, and with equally good results. He refers to the advantages of his method in infected cases, where drainage through the pouch of Douglas is easily carried out, although he believes that it is not often necessary. By keeping the uterus directed forward the pelvic cavity can be cleaned and dried much better than by any other method. P. does not advocate his method for all cases, but it is better where there are marked adhesions between the uterus and the abdominal wall; in marked cases of pendulous abdomen, and in cases with undilated cervix.

JOHN A. KYNOCH.

Case of strangulated tubo-ovarian hernia in an infant.

ARTHUR B. EUSTICE (*Journal Amer. Med. Ass.*, lxii, page 772). A coloured infant, six months old, had a rupture which the mother was usually able to reduce. It became strangulated, red, swollen, and tender, but the infant was not in great distress. The sac, on being opened, was found to contain a tube, ovary, and broad ligament. The parts were removed and the child did well. C. N. L.

REPORTS OF SOCIETIES.

ROYAL SOCIETY OF MEDICINE.

Section of Obstetrics and Gynæcology.

Meeting held Thursday, June 11th, 1914.

The President, Dr. W. S. A. Griffith, in the Chair.

Exhibition of Specimens.

Dr. Macnaughton Jones showed a twin female monster, exhibiting thoracopagus approaching to prozygosis in which the thoracic centres were united by the visceral laminæ. The twins (female) faced each other and were united above the umbilicus and equally developed. The umbilical cord was single. The union extended unusually far forwards, involving not only the thoracic wall, but also the neck, lower jaw, and lower lip, being an example of emprosthozygosis thoraco-didymus (Gurlt).

Mr. Alban Doran, in discussing the specimen, said that Schwalbe classed these very rare monsters under the type of prosopothorocopagus. Three others have been reported by Barkow, Daude and Otto. Mr. Doran considered that this type of monster was a true connecting link between the chest-united thorocopagus and the head-united prozygosis, or cephalothorocopagus twin monsters.

Remarks were also made by the President, Dr. Amand Routh, and Professor Waterston.

Mr. Gordon Luker showed a lithopædion removed from a patient six months pregnant, a 4-para, æt. 33, when six and a half months pregnant. The date of the ectopic gestation was apparently twelve years before—the first pregnancy. Pain and a lump in the right iliac region had been noted by the patient in each succeeding pregnancy. The specimen was ovoid in shape, about 6 c.m. by 4.5 c.m., covered by an elastic bony shell about 0.2 c.m. thick. Abortion occurred on the third day after operation. The patient was under the care of Mr. Frank Kidd in the London Hospital, and the operation was performed by him.

Epidiascope Demonstration.

Professor David Waterston showed a decidual cast containing a very young embryo. Professor Waterston's demonstration of this specimen and the exhibition of a wax reconstruction model of the embryo were of the greatest interest to the Section, and, as the President and others remarked, the Society owed Professor Waterston a deep debt of gratitude for bringing the specimen before their notice, not only on account of its rarity, but on account of the immense scientific value for teaching purposes as showing the process of implantation and development of the early human embryo. The clinical history was that the decidual cast was passed ten days after a missed menstrual period. Careful examination of the interior of the cast showed a small pea-like nodule which it was thought might be an embryo. It was accordingly sectioned and an embryo was found in it. Many of these sections were shown by means of the epidiascope, and Professor Waterston's demonstration of them was followed with the deepest interest. The embryo was nearly 3 mm. in length and was apparently about three weeks old. Sections showed the early embedding of the

embryo, and the characteristic features of the various positions of the decidua. A beautiful reconstruction model was also shown of the embryo, yolk sac, amnion, chorion, and decidua, greatly magnified. A complete account of the embryo and drawings will be published shortly by Professor Waterston in the *Journal of Anatomy*.

The PRESIDENT remarked that if it were possible to procure duplicates of Professor Waterston's model they would be invaluable for teaching purposes.

Dr. ARCHIBALD DONALD (Manchester) read a short paper on a case of so-called

CHRONIC METRITIS IN A NULLIPARA.

This case was of interest in that it shed some light on the ætiology of a somewhat obscure condition—the so-called chronic metritis in virgins, which seems to be a general hypertrophy of the whole uterus or that found in women who had never been pregnant, and in which, of course, subinvolution is out of the question. The patient was first seen in March, 1912. She was æt. 45, married four years, and suffered with profuse menorrhagia. There had been profuse and painful menstruation since the age of 14. Latterly the periods had lasted as long as three weeks at a time. The uterus was found enlarged to the size of a three months pregnancy. The patient was thought to have a fibroid. The patient did not improve, and owing to the profuse losses and the effect on her general condition, supra-vaginal hysterectomy was performed in May, 1913. She made a good recovery. On examination of the specimen, the uterus was regularly enlarged. There was no fibroid. The length of the uterus without the cervix was $3\frac{1}{2}$ inches, and its walls 1 to $1\frac{1}{8}$ inch thick. The endometrium was also enormously thickened and measured a little over half an inch. Microscopically, the uterine wall consisted of muscular and fibrous tissue in the same proportion as in a virgin uterus, the blood-vessels were not enlarged, and there was no elastic tissue other than the internal elastic layer of the blood-vessels. The enormously thickened endometrium consisted of glands very much enlarged in some parts, but not in others. The inter-glandular tissue was of average density and in places slightly œdematous. The blood-vessels were numerous. The enlargement of the endometrium is therefore a true hypertrophy, but its causation seemed obscure. Dr. Donald referred to the paper he had published ten years ago on " Chronic Endometritis and Chronic Metritis in Virgins."

Dr. W. FLETCHER SHAW (Manchester) read a paper on

THE SUB-DIVISIONS OF CHRONIC METRITIS.

This paper was read directly after Dr. Donald's, and by the wish of the President the two papers were discussed together. The author admitted that he was dealing with a difficult subject, but he held that chronic metritis was one of the commonest gynæcological " diagnoses," but that from a pathological standpoint the nomenclature was hardly correct. Possibly " chronic metritis " as a clinical term should be retained, by which was meant a uterus symmetrically enlarged and hard, which contained no fibroids or malignant disease, and which caused hæmorrhage, pain and leucorrhœa.

A multiparous uterus could always be distinguished from a parous uterus by the distribution of the elastic tissue which is found in the parous uterus to surround the vessels or groups of vessels. Reference was made to the work of James R. Goodall, of Montreal, on this subject, and to the

condition called "sub-involution." The persistence of the "elastic layers" round the vessels in a parous uterus was always to be found.

The author, in the course of his arguments, maintained that chronic metritis *is* a clinical entity, and includes at least two groups of pathologically different uteri. The object of the paper was to describe two of these groups based on the examination of 29 uteri removed with the diagnosis of "chronic metritis."

GROUP 1 was classified as *chronic subinvolution*, in which the uterus is regularly enlarged and hard, and the symptoms those of hæmorrhage, pain, or leucorrhœa.

Many such cases were nowadays termed "fibrosis uteri," but the author pointed out that this was an incorrect term and quite unnecessary, further it was very confusing in that there is no marked increase of fibrous tissue at any particular region, but that muscular tissue and fibrous tissue are both found enormously increased in such uteri. The most marked changes in chronic sub-involution is that the vessels and groups of vessels are found surrounded by thick "slabs" of dense elastic tissue and by muscular tissue thickly interspersed with elastic tissue. This change is very marked in cases attended with severe hæmorrhage. The blood-vessels are not necessarily thickened or enlarged, but new formed vessels are sometimes seen surrounded by the old internal elastic lamina outside which is the old vessel wall impregnated also with elastic tissue. In 15 out of 25 specimens examined the endometrium was greatly thickened, but in none was it œdematous, as it probably would have been had the thickening been due merely to congestion. The great thickening of the uterine wall itself was brought about by involution being retarded and the muscular tissue and elastic tissue not being absorbed.

GROUP 2.—*Hypertrophic uteri.*—In this group the patients had never been pregnant. The cases presented similar clinical features, and there was regular enlargement of the uterus.

Four cases of this nature were described, two were virgins and two married nulliparous women. All had severe hæmorrhage, and dysmenorrhœa, and one had been curetted three times without benefit.

Histologically these uteri differed from Group 1 and form a normal parous uterus in the arrangement of the elastic tissue. In Group 2 the elastic tissue followed the same arrangement as in a virgin uterus, but was present only as the internal elastic lamina of the vessels and a few fine fibrils in the media and adventitia and between muscle bundles of the mesometrium. None of these uteri showed an increase in the percentage of fibrous tissue found, nor were the blood vessels increased in size or number. In this group the uterine walls are very thick also, but, instead of the increase being due to want of absorption, it is produced by a definite hypertrophy of all the constituent elements.

Again, in Group 2 the endometrium was found enormously thickened and seems to be the primary fault in such cases, becoming thickened from some cause unknown and giving rise to the hæmorrhage so typical in the clinical histories. Many of these cases are cured by curetting in the early stages, while in Group 1 hysterectomy is often needed.

Reference was made to the work of Beckwith Whitehouse and Henry Briggs, but the author (Dr. Fletcher Shaw) maintained that in a large

majority of cases he had investigated, syphilis as a causative factor was out of the question.

The papers were discussed by the PRESIDENT, D. T. J. STEVENS, Dr. LOCKYER and Dr. EDEN.

Dr. DONALD and Dr. FLETCHER SHAW replied.

THE NORTH OF ENGLAND OBSTETRICAL AND GYNÆCOLOGICAL SOCIETY.

Meeting held in Sheffield, March 20, 1914.

The President, Dr. WILLETT (Liverpool) in the Chair.

Dr. DOUGAL (Manchester) exhibited a specimen of

ANGIOPAGOUS TWINS,

which appears on p. 42.

Dr. OLDFIELD (Leeds) asked how it was that the circulation of a stronger twin does not overpower that of a weaker more frequently.

Dr. DOUGAL, in reply, thought that in most cases the communication between the circulations was much less intimate and complete than in cases like that which he had described.

Dr. ARNOLD W. W. LEA (Manchester) read the notes of a case of

LARGE OVARIAN CYSTOMA CAUSING SYMPTOMS.

On April 9th, 1913, I was asked by Dr. Molloy, of Blackpool, to come and see a patient who had a large ovarian cystoma which was causing severe symptoms. He wished me to come to Blackpool on April 10th and to bring everything required for an abdominal section. I performed the operation at the patient's house. Dr. Molloy assisted. We removed a large ovarian cystoma of the left ovary which almost filled the abdomen. The tumour had torsion of the pedicle, and was also adherent to the omentum and the intestine. These were separated before the tumour was removed from the abdomen. The left Fallopian tube was a hydrosalpinx and was removed with the tumour. The uterus and right appendages were normal. All hæmorrhage being controlled, the abdominal wound was closed.

As a precaution against peritoneal infection the pouch of Douglas was incised through the vagina and was firmly packed with aseptic gauze.

The patient stood this severe operation very well and was in good condition when I left about two hours after. Dr. Molloy asked me to go over to Blackpool the next day.

I found the patient fairly well but with distension of the abdomen, which was relieved by a saline and brandy enema. After this the patient made an uneventful recovery.

Dr. CLIFFORD (Manchester) deccribed a case of

OVARIOTOMY FOLLOWED BY ECTOPIC PREGNANCY IN THE STUMP,

which appears on p. 38.

Mr. MILES PHILLIPS (Sheffield) commented on the difficulty in making out exact anatomical relationships in the specimen. He threw out a suggestion to members meeting with cases likely to lead to similar difficulties: it would be useful to attach identifying ligatures to the stumps of the round and ovarian ligaments, at the time of the operation, when these structures could usually be identified.

Dr. GREY NEWTON (Sheffield) asked if any healthy ovarian tissue had been found in the cystic ovary.

The PRESIDENT had once before seen a similar case in which the gestation sac was interstitial. The patient refused treatment for three months and died from rupture of the sac before operation could be performed.

Dr. CLIFFORD replied that there was some healthy tissue in the hilum of the ovary. The route of the ovum was an interesting question, most of the round ligament had been removed at the previous operation, and what remained of it was not recognisable at the second operation.

Mr. MILES H .PHILLIPS (Sheffield) described a case of

DOUBLE OVARIAN CYST,

which appears on p. 45.

Mr. R. FAVELL (Sheffield) thought the Trendelenburg position was to blame for accidents of this kind. He remembered a case in which a patient was opened and examined for the cause of intestinal obstruction. Nothing was found, the abdomen was closed and the patient died. An ovarian tumour with a long pedicle was found in the splenic region causing the obstruction.

Mr. PHILLIPS, in reply, said that Dr. Gemmell had advised him always to remove both ovaries in old women, as he had left one in a patient aged 80, who came back with another ovarian cyst at the age of 85.

Dr. GREY NEWTON (Sheffield) described

AN OVARIAN TUMOUR.

Mrs. B., æt. 44 years, 8-para. Menses regular, general health good. Came to out-patients last August, 1913, complaining of a swelling and pain in lower part of body. On examination I found a cystic mass—size of a large fœtal head—on the right side. I advised her to come in, but she didn't turn up till last month, February, 1914, when she was brought up in the ambulance.

The house surgeon saw her on admittance, and from her general appearance derived the impression that she was suffering from either heart or kidney disease. She had great dyspnœa, abdomen full of free fluid, heart displaced to left owing to effusion in her right pleural cavity extending nearly to level of 3rd rib. There was no albumen in the urine, nor could any very definite murmur be detected over any valves of the heart. The effusion to the extent of 1½ pints was drawn off.

It was reported pathologically as being a simple passive effusion. A few days after the aspiration I did a laparotomy, when a great quantity of straw coloured fluid gushed out, and I was afterwards able to withdraw an ovarian cyst from the right side, the size of a large fœtal head.

There were no adhesions except a portion of the omentum which was adherent to the parietal peritoneum; this felt hard so I cut a portion out for a report—which was returned as not malignant. The ovarian cyst was reported to be a cystadenoma.

The patient felt much better after the removal of tumour and fluid and continued to make satisfactory progress for a fortnight. The effusion in her chest reformed, and she had to be aspirated on three occasions. Two hours after the last aspiration she suddenly died. Unfortunately no *post-mortem* was allowed. Clinically this case pointed strongly of a malignant character, but the pathological report negatived this view.

Mr. W. W. KING said that, from the examination of the sections, he was of opinion that the tumour was malignant, and that he thought that the clinical history supported this view.

Mr. FAVELL (Sheffield) exhibited

A LARGE FIBROMA OF THE UTERUS UNDERGOING SARCOMATOUS DEGENERATION.

The patient from which the uterus was removed was 47 years of age, had been married 23 years, and never pregnant. Her menstruation had been regular up to two years ago, her periods never lasted more than two days and were scanty in amount, never any menorrhagia. For the last two years she had been irregular, being unwell only every three or four months. She had noticed the swelling increasing in size during the last twelve months. At the time of the operation it reached four inches above the umbilicus. There were several subperitoneal fibromata. Dr. King. who furnished the pathological report, considered it a myoma undergoing sarcomatous transformation, the tumour being mainly composed of myomatous tissue undergoing hyaline degeneration, but in places the cells are short and closely packed together, while the nuclei are large and irregular in shape. The tumour is also very vascular and contains numerous thin-walled blood vessels.

Dr. FOTHERGILL (Manchester) read a short paper on

A FURTHER MODIFICATION OF THE TECHNIQUE OF ANTERIOR COLPORRHAPHY.

which appears on p. 29.

Mr. MILES PHILLIPS was glad of the opportunity of praising Dr. Fothergill's method of operating on cases of procidentia with large cystoceles. It had invariably given him most satisfactory results. It was always gratifying to use a procedure based on sound theoretical considerations, especially if it gave good results. He was not sure that he appreciated Dr. Fothergill's new suggestion, as it seemed to him likely to be more difficult to dissect off the vaginal wall from the urethra towards the cervix than to do this in the opposite direction. Still it was probably a neater method, and he should certainly give it a trial.

Dr. STOOKES (Liverpool) had done two cases during the last week and noticed that the cervix disappeared into the hollow of the sacrum. In suturing he would put a buried suture or two in front of the cervix.

In reply to Dr. Stookes and Mr. Miles Phillips, Dr. FOTHERGILL said that he had tried the use of buried sutures for bringing together the more central portions of the parametric tissue in front of the cervix. He had also used big sutures passing through the margins of the wound at its widest part and also through the ânterior portion of the cervix. He had, however, found that these were unnecessary, and he now began stitching from behind forwards until the stump of the cervix was surrounded by vaginal wall and completed the closure of the wound in the middle line by sutures uniting simply the cut edges of the vaginal wall and the tissues immediately subjacent to it.

Mr. W. W. KING (Sheffield) read a paper on

THREE CASES IN WHICH AN EARLY TWIN OVUM WAS DELIVERED TOGETHER WITH A FULL TERM CHILD,

which appears on p. 39.

Mr. PHILLIPS (Sheffield) was grateful to Dr. King for describing and elucidating these specimens. A correct understanding of such structures might prevent a good deal of heart-burning and even legal proceedings. It was interesting to note that the three specimens had been obtained in the reverse order of the ease with which they could be deciphered. He personally thought they were due to the early death of one of homologous twins, possibly as the result of localised disease of the uterine mucous membrane.

NORTH OF ENGLAND OBSTETRICAL AND GYNÆCOLOGICAL SOCIETY.

Meeting held in Manchester on Friday, April 17th, 1914.

The President, Dr. Willett (Liverpool) in the Chair.

Present: 20 members.

Dr. Mackay (Southport) exhibited:

Post-mortem Specimen of Ruptured Tubal Pregnancy: Symptoms suggestng Irritant Poisoning.

which appears on p. 36.

The President said they must thank Dr. Mackay for bringing this unusual case before the Society, unusual both in the rapidity of the fatal termination and in the site of rupture. He believed Dr. Cuthbert Lockyer described a case which was rapidly fatal, but he had never seen one fatal in 5 hours. The question was whether death was due to shock or hæmorrhage.

Dr. Fothergill (Manchester) exhibited a specimen of

Endothelioma of the Ovary.

Dr. Fothergill showed a multilobular solid tumour of the ovary which proved on microscopic section to be an endothelioma. The patient was a woman, aged 34, who had never been pregnant. The only symptom complained of was gradual enlargement of the abdomen. The menstrual periods were regular, 28 days type and 1 day duration. On examination a large firm tumour occupied the lower part of the abdomen and reached up to the umbilicus. It felt like a mass of fibroids or multilocular ovarian cystic growth. On vaginal examination it could not be definitely distinguished from the uterus. It was found to be developed from the left ovary, and was removed on March 8th with the corresponding tube. The patient was discharged well on March 30th. Sections presented the structure which some years ago would have been called myxo-sarcoma. There were large areas of myxomatous tissue, but scattered through these were compact cellular areas composed of large cells arranged round the lumina of small blood vessels from whose endothelium the cells of the new growth were evidently derived.

Dr. Oldfield (Leeds) exhibited

Four Specimens of Mesothelioma.

1. Female, aged 60, with abdominal tumour as large as full term pregnancy: said to have been fibroid 20 years ago. Water-logged from cardiac disease and flooding for 2 months. Diagnosed as sloughing fibroid: total hysterectomy, with good recovery and relief of cardiac symptoms. Pathologist reports specimen as mesothelioma.

2. Female, aged 40, with solid pelvic tumour and mass across middle of abdomen. She has pain, wasting and drenching sweats. Masses removed consisted of uterus with growth in endometrium, solid tumours of both ovaries, retrocœcal mass, the whole of great omentum infiltrated with masses of growth, and a portion of anterior abdominal wall. Patient remains well with no sign of recurrence 6 months after operation. Pathologist reports specimen as mesothelioma.

3. Female, aged 35, with solid tumour in lower left abdomen: thought to be fibroid, but found to be solid ovarian. Pathologist reported specimen to be sarcoma first of all, then carcinoma, and later decided that it is mesothelioma.

4. Female, aged 35, with hæmaturia and enlarged kidney. Growth of kidney found and nephrectomy done for it. In some parts sections look like sarcoma and in others like carcinoma. Diagnosis mesothelioma operation 2½ years ago and no recurrence.

These 4 cases and 2 others in Dr. Oldfield's practice during the last 3 years lead him to think that mesothelioma must often be less malignant than carcinoma or sarcoma of the uro-genital organs.

Dr. FLETCHER SHAW (Manchester) said they must all be struck with the large numbers of these tumours which had recently been published. This could not mean that they did not occur formerly, but that they were not recognised. Many of these specimens presented appearances, histologically, very like carcinomata and no doubt were formerly classified as such. In fact, in the 1900 edition of Coats' Pathology they were so classified on account of their appearance, though he recognised that they should, strictly speaking, be called sarcomata. Another point of interest in these tumours was their apparently low malignancy. Dr. Shaw did not like the term "mesothelioma," used apparently because these tumours arose from mesoblast. It seemed much better to classify all these tumours as "sarcomata." using "endothelioma" as a section of this large class and "perithelioma" as a subsection of "endothelioma."

Dr. FOTHERGILL and Dr. DOUGAL (Manchester) showed a specimen of

ANEURYSM OF THE INTERNAL ILIAC ARTERY,

which appears on p. 32.

Dr. OLDFIELD (Leeds) said it was most important to know whether the cause was pathological or traumatic: whether the patient had had syphilis or whether forceps had been used at the previous confinement. In his opinion it seemed likely that the cause was traumatic acting in a syphilitic patient.

Dr. FOTHERGILL, in reply, said this was the patient's first child, and he had delivered her by Cæsarean section: there was no history of syphilis.

Dr. DOUGAL said there was no evidence of endarteritis.

Dr. WALLS (Manchester) exhibited a specimen of

CÆSAREAN SECTION AND WERTHEIM'S HYSTERECTOMY FOR PREGNANCY WITH CARCINOMA OF THE CERVIX AND DOUBLE PAPILLOMATOUS OVARIAN CYSTS.

The patient, H.T., a 6-para, aged 41, was admitted to St. Mary's Hospital on February 3rd, 1914. She was then 6½ months pregnant, and had suffered considerable irregular hæmorrhages for 3½ months.

Abdominal examination shewed a uterus enlarged to correspond with the duration of pregnancy, and closely connected with it was a rounded swelling about the size of a fœtal head and extending to the left hypochondrium. This was thought to be a soft fibroid, from its close connection with the uterus. Vaginal examination shewed the cervix to be very hard and rigid, bleeding fairly freely after being touched. It was freely mobile and the vaginal roof was healthy.

A microscopic section was made of a snip from the cervix and proved to be definitely columnar carcinoma. On Feb. 20th, 1914, when the pregnancy had extended just over 7 months, Cæsarean section was performed—a well-formed child of 5 lbs. weight being born alive—after which two ovarian cysts and the uterus were removed *en masse* by Wertheim Panhysterectomy—and one large gland was removed from the front of the left external iliac artery.

The child unfortunately died of hæmatemesis six hours after birth: but the mother did well and was discharged three weeks after operation.

The specimen (reported upon by Dr. Shaw, who prepared the slides afterwards mentioned) comprises the uterus regularly enlarged from recent pregnancy, and presenting the longitudinal incision of Cæsarean section, but the *body* shewed no evidence of fibroids or other tumour. The *cervix* was considerably thickened and friable, and a microscopical section shown proved this to be due to columnar carcinoma.

Both *ovaries* were cystic, the left being the size of a fœtal head, the right of an orange: in the fluid content of these cysts were large quantities of beautifully fine papillæ: and one ovary alone appeared to have any normal ovarian tissue as judged by the naked eye, and that was only the size of a pea.

Sections of the cyst walls shewed them to consist of fibro-muscular tissue lined by branching papillæ covered by columnar cells. No evidence of carcinoma was found in the ovaries.

Dr. OLDFIELD (Leeds) asked if the Wertheim's operation was easier because of the pregnancy.

The PRESIDENT said he had recorded a case of Cæsarean section and hysterectomy for an 8 months' pregnancy with carcinoma of the cervix. A complete Wertheim's pelvic dissection was deemed inadvisable, as the patient was not in a condition to stand a prolonged operation, but the patient was reported well five years afterwards. The operation was very easy.

In reply to remarks of Drs. Oldfield and Willett, Dr. WALLS described the operation as having been a much easier one to perform than was usually the case where there was no pregnancy.

Dr. OLDFIELD (Leeds) read the notes of

THREE CASES OF OVARIAN TUMOUR OBSTRUCTING LABOUR.

1. Primipara, aged 25, in labour 48 hours, very ill: uterus in continuous action. Delivered by Cæsarean section: child markedly compressed and in extremis. Heart beat only 10 per min. on delivery. Tumour removed 2 weeks later. Mother and child did well.

2. Multipara, aged 42, in labour 16 hours. General condition good. Abdomen opened, uterus turned out of abdomen and tumour extracted and removed. Uterus replaced and abdomen closed. Head now found at outlet and readily extracted with forceps. Mother and child did well.

3. Primipara, aged 38, 7 months pregnant. Cord prolapsed and pulsating: child lying transversely. Tumour easily pushed up out of pelvis; child turned and extracted after coming head being perforated. Mother made good recovery.

The three cases exemplify the three common methods of dealing with this complication of labour.

Dr. CLIFFORD (Manchester) described a case of

CÆSAREAN SECTION FOR CONCEALED ACCIDENTAL HÆMORRHAGE.

The patient, aged 39, at full term in her eleventh pregnancy, was admitted to St. Mary's Hospital, Manchester, on March 3rd, 1914, in a state of collapse, with a very feeble pulse of 136 and a temperature of 96.6° F. She was pale and restless. The abdomen was distended, the uterus tense; no fœtal parts could be felt and the fœtal heart could not be heard. There were no signs of uterine contractions. The os admitted two fingers, and there was no hæmorrhage from the vagina.

Shortly after admission the pulse could not be felt at all and the patient became more collapsed in spite of the infusion of saline. It was therefore decided to operate. Cæsarean section followed by supra-vaginal hysterec-

tomy was rapidly performed. An interesting point noted was that there was no hæmorrhage from either the abdominal or uterine wall when they were incised. On opening the uterus blood, under very considerable pressure, spurted out and there were two large clots behind the placenta which had been situated on the posterior wall.

After the operation the pulse rapidly improved and the patient made a good recovery. The convalescence was without incident except that three weeks after the operation the temperature rose to 101° F. for no obvious reason and then gradually returned to normal.

There were large dilated veins in the left broad ligament that looked ready to rupture if they had been stretched a little more, as happened in a case recently reported by me.

Dr. WALLS (Manchester) said Cæsarean section followed by hysterectomy was the best treatment for concealed accidental hæmorrhage. In the old days many of these correct treatment had been carried out.

MIDLAND OBSTETRICAL AND GYNÆCOLOGICAL SOCIETY.

Meeting held at the University, Bristol, on Tuesday, April 7th, 1914.

Dr. W. H. NEWNHAM in the Chair.

Mr. CHRISTOPHER MARTIN showed the following specimens :—

1. DOUBLE FIBROMATA OF THE OVARY WITH SARCOMATOUS DEGENERATION.
2. HYPERTROPHY OF THE CLITORIS ASSOCIATED WITH PRURITUS VULVÆ.

Mr. BECKWITH WHITEHOUSE was of opinion that the Neoplasm described as " Sarcomatous Degeneration of a Fibromyoma " was a mixed celled Sarcoma. In the section shown the tissue was highly cellular and fibrous tissue was present in a minimum amount. He believed that the majority of such tumours were true sarcomata and he did not agree with the suggestion that sarcomatous change might occur in a pure fibroma. In all similar cases he thought it most important that sections should be taken from different aspects of the tumour, as a definite opinion could not be expressed upon the one section shown.

Professor WALKER HALL agreed with the previous speaker on the importance of examining several sections from tumours of doubtful pathology such as the one shown. Mr. Martin's specimen was undoubtedly malignant, but he thought that from the diversity of the cells present the tumour might well be classed under the category of " Teratomata." He much objected to the term " Sarcomatous Degeneration." The malignant change was in no sense of the word a degeneration.

MYXOMATOUS TUMOUR OF BLADDER.

Mr. BECKWITH WHITEHOUSE (on behalf of Dr. Thomas Wilson) showed specimens of a myxomatous neoplasm growing from the sub-peritoneal tissues over the bladder, and the uterus from the same patient containing a malignant neoplasm of obscure nature

The specimens were obtained from a multipara, aged 54, complaining of uterine hæmorrhage and abdominal swelling of 8½ months' duration. At operation the abdominal wall was found to be infiltrated with glairy mucinous material which communicated with a cystic neoplasm growing in the sub-peritoneal tissues over the fundus of the bladder. This cyst contained the same tenacious material and three spontaneous perforations were present in its wall. The growth did not communicate with the interior of the cavity of the viscus but partial cystectomy was required for

its complete removal. The uterus appeared to be uniformly enlarged and was therefore removed together with both its appendages. It was found to contain a sessile polypoid tumour arising from the fundus and posterior wall and projecting into its cavity. The distal portion was hæmorrhagic and on section it was soft and diffuse. Free blood clot was present in the uterine cavity. Microscopical sections taken from different parts of the tumour showed tissue composed of oval, rounded, and cubical cells. The former appeared to be sarcomatous; the latter, however, were in many cases arranged in narrow alveoli and columns and apparently were of carcinomatous nature.

Professor WALKER HALL thought that the uterine tumour was entirely carcinomatous in nature and that the vesical growth was a metastasis which had undergone mucoid degeneration. In the sections shown of the latter, malignant cells were present in the wall of the cyst which had a close resemblance to those forming the alveoli of the uterine tumour.

Dr. EDGE observed that the microscopical appearances of the two tumours were so entirely different that it was difficult to imagine that they both had a common cause.

HÆMORRHAGE INTO THE OVARY AS A CAUSE OF DYSMENORRHŒA.

Mr. BECKWITH WHITEHOUSE read a paper upon Dysmenorrhœa associated with Petechial Hæmorrhage in the Ovary, which will appear in full in the *Journal*.

Mr. MARTIN asked if the character of the pain had any relation to its seat in the ovary and whether this condition would explain the "Mittelsmerz" described by German writers. He also asked for information regarding any sign or symptom which would help in diagnosing similar cases.

Professor WALKER HALL said that the paper had raised many important points concerning the pathology of ovarian hæmatomata. It was of course a difficult point to ascertain how far small hæmorrhages into the organ were physiological and until this was settled deductions as to pathology could not be made. He referred to the relationship of corpora albicantia with hæmorrhage and thought that the majority of these bodies were due to the organisation of blood clot and represented physiological hæmorrhage into the stroma.

Dr. EDGE thought that the removal of the uterus and its appendages, in the absence of gross disease, was very radical treatment for such a symptom as dysmenorrhœa.

Dr. NEWNHAM agreed with the statement of the previous speaker and said that if Mr. Whitehouse's views were correct in attributing the pain in some cases to the ovaries, a considerable advance in the treatment of this condition had been made, particularly if it was possible to locate from the character of the pain the seat of the lesion.

PRIMARY CARCINOMA OF THE URETHRA.

Dr. EDGE read a short communication upon Primary Carcinoma of the Female Urethra based upon three cases which had come directly under his own observation. This paper will be published elsewhere.

Mr. BECKWITH WHITEHOUSE thanked Dr. Edge for bringing these three very interesting cases forward. A few years ago, in a paper communicated to the Obstetrical and Gynæcological Section of the Royal Society of Medicine, he had drawn attention to the same subject and had collected all the cases then recorded. Since the publication of this paper several cases had come under his notice, but so far he had no reason to change his

views from the opinions there set forth. The condition, although uncommon, was not so rare as had been thought. The vulvo-urethral growths were by far the most common.

Mr. RAYNER referred to a case that had been under his care in which the growth involved the lower quarter of the urethral canal. No glands were present and he had excised the lower half of the canal. The tumour was a squamous epithelioma. The chief symptoms had been severe pain and hæmorrhage on micturition.

EDINBURGH OBSTETRICAL SOCIETY.

Meeting held on March 11th, 1914.

Sir HALLIDAY CROOM, President, in the Chair.

Dr. J. W. BALLANTYNE read a paper on

STILL BIRTHS REGISTRATION.

in which he pointed out the extraordinary diversity of practice which existed in different parts of the world and even in different parts of the British Empire with regard to the registration of dead-born and still-born babies and in respect of the definitions of live- dead- and still-birth accepted for official use in these different places. There was a veritable jungle of legislative, official, and lexicographic divergencies of theory and practice. He was of opinion that the jungle had been entered by not following the straight path of the study of antenatal and intranatal physiology, and he thought that the only way out was to investigate and to re-state the conditions of life and death before and during birth. The word *still-birth*, for instance, should not be regarded as synonymous with dead-birth, but should be reserved for those cases in which there was a hesitancy in the smooth passage of antenatal into postnatal life; whilst not having put off the characters of the life before birth the child showed a delayed putting on of these which marked life after birth. A still-born infant might be resuscitated or " trans-animated " (as he proposed to call it), and therefore it could not have been dead. He pleaded for a revision of the terminology of the subject before a Still-births Registration Bill stereotyped the definitions for ever. It was really an Antenatal Deaths Registration Bill which was called for.

Meeting on May 13th, 1914.

Sir HALLIDAY CROOM, President, in the Chair.

A discussion was held on Dr. J. W. BALLANTYNE'S paper on

STILL-BIRTHS REGISTRATION.

At the outset, Dr. BALLANTYNE said that his paper was really a consideration of terminology, but the terms discussed had a practical bearing and not merely an academic interest. The difficulty was with the terms " still-birth " and " dead-birth " and with their equivalents in foreign languages. In their use there was great confusion. The great problem standing in the way of definition was that, in many cases, one had to wait before being sure that any given birth was going to be a live-birth or not. His explanation of the confusion was (1) a lack of adequate recognition of the physiology of antenatal life, and of the changes which occur or may occur at birth, and (2) the habit which the public with the connivance of the profession and the profession itself in some places had got into regarding still-

birth as a synonym of dead-birth. As Parliament was soon to bring in legislation dealing with still-births and their registration, it seemed well to discuss the terminology before the words still-birth, dead-birth, live-birth, etc., were finally crystallised along erroneous lines. The definitions he had drawn up had been unanimously adopted by the Council of the Society.

Dr. LESLIE MACKENZIE (Local Government Board) felt that three things hung together, the notification of births, the proper regulation of midwives, and the whole question of infantile mortality. When the notification of births became universal it would mark an advance. The Local Government Board had practically decided where the Act was not adopted, to have it done by themselves. He thought that, along these lines, the providing of proper training of midwives in connection with the Midwife's Act, would be good. It should include definite and special teaching in such aspects of the pathology of still-births as were desirable. He had some doubt as to whether the distinctions which Dr. Ballantyne had made, though scientifically necessary, were of practical value. In spite of the risk of comparison, however, he thought that a vast amount of useful information could be got, even though some considered still-birth one thing, live-birth another thing, and dead-birth another thing, while others did not differentiate sufficiently between them. He thought that even a limited amount of information, in the hands of good men, such as the Local Authorities of Glasgow and Edinburgh, would be of great value. If the Registrar-General had to distinguish 5 forms of tuberculosis and 2 or 3 forms of pneumonia, it was not asking very much for medical men to make these distinctions as regards births.

Dr. J. C. DUNLOP (Registrar-General's Department) agreed with Dr. Ballantyne as regards the general lines he had laid down. He considered, however, that notification, not registration, was the best method of acquiring and coordinating the information, because registration was not primarily an administrative machine for the production of vital statistics. These were a by-product. Registration was done by men with no medical knowledge and there was no medical supervision of this machine until it was too late to make any enquiries which might be necessary under these circumstances. In regard to notification there was complete medical supervision. The registration of these cases, also, did not fit in with the established system in Scotland. There were three registrations,marriages, deaths, births. The birth register only dealt with children that live in order that they might have, when they grew up, their civil rights established. If one introduced other registers for miscarriages, dead-births and still-births, he feared the ordinary system of registration would be upset. He would suggest three considerations why the medical officer should be directly notified :—(1) The information must be secret, (2) the information is passed on to a man with the technical knowledge necessary to appreciate it, and (3) it in no way complicates the present system.

Professor HARRY LITTLEJOHN thought that the chief function of statistics was not purely civil. Registration had been influential in the direction of preventative medicine. He thought that Dr. Ballantyne's definitions might be simplified. They wanted to know, firstly, the existence of abortions and dead-born children. Let there be a general notification of dead-born children, whether they died some weeks before or during birth. This admittedly did not give all the information they wanted. But how were they to discover, in ordinary cases, whether a child died of syphilis, from the cord being round the neck, or from other birth causes? Secondly, they

wanted a record of live-born children. He doubted if still-birth was a useful definition. Many causes might produce it. If it became alive it should be called live-born.

Dr. BERRY HART thought that we could not limit the sphere of registration to its civil value. It would give information regarding the cause of fœtal death, syphilis, rickets, etc. He agreed that it was doubtful if the introduction of still-birth would be of any value. If a child lived only a few minutes, it should be classed as dead-born; if it was still-born and lived for days it was a live-born child. As to abortions, he thought the statistics would be worthless.

Dr. KEPPIE PATERSON thought that "Still-Births Notification" would cover the case better. In maternity hospitals and dispensary practice the information would be valuable; not so as regards private practice generally. Many dead-births were syphilitic, but the majority of those giving evidence before the Commission on Venereal Diseases were against compulsory notification. If the doctor were compelled to notify an abortion or dead-birth as syphilitic, there would be trouble. The husband would deny it and would refuse a Wassermann test. And if the cause of dead-births was not put in, where was the value of the statistics?

Dr. FREELAND BARBOUR thought two classes of cases had to be distinguished: those in which the child died *in utero* and was expelled subsequently, at what time it did not matter, and those in which the child died during the process of labour.

Dr. JAMES RITCHIE advocated the compulsory notification of births after 28 weeks' gestation.

Dr. HAIG FERGUSON thought that the idea of "still-birth" was so firmly established in the popular belief that for practical purposes it would be well to retain it.

Dr. BALLANTYNE, in reply, thought that notification might be the better method, but it would require serious consideration. He agreed it would be well if one could simplify the classification to include only dead-birth and live-birth, but there was a danger of losing scientific precision if one simplified too much.

Meeting held on June 10th, 1914.

Sir HALLIDAY CROOM, President, in the Chair.

Dr. A. C. MCMASTER showed specimens of

HYDATID MOLE,

in which the size of the uterus was the same as, or was smaller than, a normal pregnancy of the same age. He maintained that errors in diagnosis were apt to arise because of the usual belief that a hydatid mole was always associated with excessive expansion of the uterus. This might be so, but it was usually not the case.

Dr. FORDYCE read a paper on

A CASE OF PREGNANCY IN A MALFORMED UTERUS.

The patient was seen when three months pregnant with a swelling in the right iliac region. Bimanually it was felt to press low down into the vaginal fornix, the uterus being displaced to the right side. The diagnosis rested between tubal pregnancy, pregnancy plus a cyst in the broad ligament and pregnancy in a malformed uterus. She was left for some time—until $4\frac{1}{2}$ months—when she was operated on. The uterus was removed by supravaginal hysterectomy, some difficulty being met with on the right side owing to the opening up of the broad ligament. On cutting through the

cervix, it was found to have a double canal. The vagina and vaginal portion of the cervix were simple, therefore the condition was uterus subseptus.

Dr. BALLANTYNE thought the best treatment had been carried out.

Dr. BERRY HART read a paper on

ATYPICAL MALE AND FEMALE SEX-ENSEMBLE (HERMAPHRODITISM AND PSEUDOHERMAPHODITISM).

In some preliminary remarks the speaker stated that he held the sex-gland to be the only criterion of sex. Thus in typical and atypical cases the sex was either male or female. What one studied really was the sex-ensemble, the " altogether " of effective sex. In the normal female the sex-ensemble was made up of (1) ovary, tubes, uterus, vagina and external genitals—the potent portion, (2) the opposite sex-duct elements, epoöphoron and paroöphoron—the non-potent portion, (3) the secondary sexual characteristics as to hair distribution, mammæ, pelvis, bones, larynx and psycho-sexual powers. The normal male-ensemble had (1) descended testis, vas deferens and phallus—potent organs, (2) opposite sex-duct elements, hydatid testis (Müllerian) and prostatic utricle (hymeneal)—non-potent, (3) secondary sexual traits. In both of these the potent organs were at a maximum and the non-potent at a minimum with the secondary sex traits congruent. This was an ultimate probability result and any increase in the minimum gave a diminished maximum and a non-congruence of the secondary characters.

The essence of atypical male and female sex-ensemble was thus a ratio-disturbance in the duct elements of the sex-ensemble and secondary traits. The classification Dr. Berry Hart proposed was 1, *Atypical female sex-ensemble*, and 2, *Atypical male sex-ensemble*. There could be no glandular hermaphroditism as no ovo-testis had as yet been demonstrated. It was theoretically possible. Transverse and lateral hermaphroditism were non-existent if one took the sex-gland and not the duct elements as the criterion of sex.

I. *Atypical female sex-ensemble*. Tibiger of Copenhagen had written comprehensively on this division and Dr. Hart showed, through the kindness of Mr. John Fraser and Dr. Carnegie Dickson, a case in a six-weeks child. Such cases were extremely like hypospadic males and a prostate, uterus, tubes, ovary and vagina were present, the last opening into the prostatic sinus by slit-like vasa deferentia in the male. The secondary characters varied: the thyroid cartilages might have male or female ossification. Tibiger's second case had a wife and three children (by another father according to the widow's statement) and he had female psycho-sexual feeling. In all these cases it was significant that the suprarenals were large and this was well shown in Fraser and Dickson's case.

II. *Atypical male sex-ensemble*. This is to the atypical female division as 10 : 1. Typical cases were described by Leopold, Neugebauer, Luksch, and Tuffier and Laponite. In such the testes may be in the split scrotum. Tuffier's case was remarkable as the patient was somatically an attractive woman. The testes were in the split scrotum and this was absolutely proved by an incision, inspection of the sex gland and microscopic examination. Psycho-sexually the case was an " invert " and proposed marriage with a man. Somatically the limbs, hands, and feet were male, the pelvis female. There is thus in atypical male and female sex-ensemble a disturbed ratio between the potent and non-potent elements of the sex-ensemble duct-tract and a non-congruent distribution of the secondary

sex traits. In conclusion the mechanism of this and questions of diagnosis and prognosis and of treatment were considered. An epidiascopic demonstration of specimens was given and Mr. John Fraser's and Dr. Carnegie Dickson's specimen was shown.

Dr. BALLANTYNE supported Dr. Hart as regards terminology. He had not, however, referred to the combined ovo-testis that had been demonstrated in some cases. If there was no true hermaphroditism of course the word pseudohermaphroditism would have to go. He would prefer an English word to express "sex-ensemble" and would suggest "sex-totality." The enlargement of the suprarenals was a difficulty. It had been shown that glands outside those usually associated with sex were bound up with the sex-function.

Dr. FORDYCE thought that the term "pseudohermaphrodite" might still be retained.

Dr. JAMES YOUNG referred to a case he had seen of atypical male sex-ensemble. This individual was brought up as a girl and was married to a man. The figure and development was that of a man, but there was a vagina with a split scrotum. The testicles were in the labia. When the true state of affairs was recognised the long hair was cut short, and the female clothes were exchanged for male attire. Later the first marriage was annulled and marriage was contracted with a girl.